MAY 12 1925 Medical Lib.

VOL. LIV. MAY, 1925 No. 3.

ARCHIVES
OF
OPHTHALMOLOGY

FOUNDED IN 1869 BY HERMAN KNAPP

EDITED BY

ARNOLD KNAPP AND WARD A. HOLDEN

NEW YORK

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ARCHIVES OF OPHTHALMOLOGY.

EPITHELIAL DYSTROPHY AND RECURRENT EROSION OF THE CORNEA AS SEEN WITH THE SLIT-LAMP.

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(With four illustrations on Text-Plates VIII. and IX.)

CASES that are known by one of these names, or that may be called vesicular keratitis, disjunction of the corneal epithelium, or intermittent vesicular keratitis neuralgica are not very common, but still common enough to come within the yearly practice of many ophthalmologists. From certain findings with the slit-lamp, I believe that mild forms of this condition are much more common than has been supposed. While differing in their clinical pictures as regards the presence or absence of distinct vesicles, and their size, and the presence or absence of the acute pain often described, it would seem that all these cases have the same primary origin, and form a group which the name suggested by Fuchs for his series of cases, Epithelial Dystrophy of the Cornea, probably best describes. Of the form of this condition usually called Recurrent Erosion of the Cornea, I will quote three typical cases.

CASE I.—Mrs. T., aged forty-four, was first seen four years ago. She complained of attacks of poor vision for the past four years. Vision was $\frac{20}{50}$ and $\frac{20}{40}$. A chronic cyclitis was present in the right eye, as shown by deposits on Descemet's and slight discoloration of the iris. The lower quadrant of the cornea was distinctly rough, though no definite bleb was noted. Yellow ointment massage was prescribed,

in addition to treatment for the cyclitis, and the removal of a pair of infected tonsils was advised. In spite of this, the roughness of the cornea continued, and 6 months later, a distinct bleb *4mm* in diameter, was seen in the lower half of the right cornea. The loose epithelium was peeled off, the surface curetted, and trichloracetic acid applied. Healing was prompt, and the new epithelium appeared smooth. During the next year she suffered with numerous attacks of acute pain in the eye, and at each visit following these attacks the epithelium was seen to be raised and loosened at the same site. It was removed several times, and the surface cauterized once lightly with the actual cautery. Several times a staining area was seen above the edematous area, where the bleb had ruptured spontaneously. The pain was usually relieved when the bleb ruptured. Cataracts were developing in both eyes, and due to rapid swelling of the lens in the right eye, the tension which had been normal, became much increased necessitating a Curran operation. (Dr. H. Gifford.) After this the tension remained between 13 and 17 (Schiötz), and in March, 1922, the cataract was extracted by Dr. H. Gifford, giving vision of $\frac{5}{8}$ with correction. No more blebs developed after the Curran operation, and at a recent examination, the epithelium was seen to be smoothly and firmly attached, a superficial scar marking the former site of the blebs. The tension remained normal. The left eye never showed disturbance of the epithelium, and the cataract in that eye is progressing in the usual way, without increase of tension.

Case 2.—Mr. K., aged 32, came in complaining of pain in the left eye for the past two days. Vision was right eye $\frac{5}{10}$, left eye $\frac{5}{10}-$. The left cornea showed a slightly depressed area below the center with a gray border, resembling the scar of an old ulcer. The surface did not stain grossly, but was rough. There was a good deal of congestion, and a chronic blepharitis and meibomitis in both eyes. The right cornea was normal. Treatment for the conjunctiva and lids was prescribed. Three months later the condition was about the same. Different collyria were used for the lids, with very little result. The congestion and a sticky secretion continued and the surface of the depressed area remained rough, though subjective symptoms varied greatly. After five more months, when he was first observed with the slit-lamp after staining with fluorescin, a faint staining was seen over the whole half of the left cornea and outside this area over most of the cornea fine green staining dots of various size but all very small were seen, which retained the stain a long while. (See Fig. I.) The right eye, which was

congested but which had not been complained of, showed a similar profusion of fine staining dots all over the cornea, but more numerous in the central part. A series of milk injections was tried, with little or no effect. Three months later, when the original depressed area was still seen to stain faintly, it was found that the epithelium over it and a large area around it, was loose and could be peeled off easily. iodine was used on the denuded surface. Two days later the epithelium was almost replaced but this new epithelium was also loose and was peeled off, liquified trichloracetic acid being this time applied. After this the eye improved and felt comfortable but after another two months the pain recurred, this time also in the right eye. In this eye no staining with fluorescine could be seen by the naked eye, but the slit-lamp showed the cornea to be fairly peppered with fine green dots. Dionin was now being used and seemed to give more relief than anything which was tried. At a visit 1½ years after his first appearance, there was no staining area to be seen in the right eye, and the left eye showed only a few. There were, however, numerous fine gray spots, evidently the remains of the punctate erosions. A month later, both corneas were again covered with fine staining areas. At no time were any of these spots on the right cornea large enough to be seen with the naked eye, and the cornea had a normal luster and light reflex. Recently, two years after his first visit, the right eye is not congested but still shows numerous punctate staining areas over some of which the epithelium is present but elevated. The left eye was much congested and showed practically the same condition as the first except that the central opacity is now considerably denser. Quinine is now being tried internally.

Case 3.—Mrs. M., aged 38, a high myope with immature cataracts, had showed nothing abnormal about either cornea during over a year's observation, until a preliminary discission was done on the left eye. Immediately after this, a severe edema of both lids occurred and a small raised area of epithelium was noted below the center of the cornea. This remained small and had apparently disappeared at the time of her corneal expression four days later. At the operation, part of the nucleus slipped behind the iris and was left to absorb. The same small area was noted during healing. There was no untoward reaction till two weeks later, when the patient appeared with high tension and the remnant of nucleus jammed in the pupil. This was extracted with the loop with some difficulty but without loss of vitreous. The reaction was not marked, but the cornea showed striate keratitis and a great part of it was rough for a week. A week

later the patient appeared, stating that on awaking at night and opening her eyes, she felt a severe pain in the left eye which had remained very painful since. The whole central third of the cornea was seen to be raised in a distinct bleb. This was peeled off easily with forceps, when it was found that the epithelium for several millimeters around was also loose and this was also removed. The surface was touched with tincture of iodine and a double bandage applied for five days. At the end of that time, the cornea was smooth except for a small roughened area above the center. Numerous attacks have occurred since with intense pain, photophobia and edema of the lids and at each time larger or small blebs were present. The epithelium was peeled off several times and the surface painted with fresh aqua chlori. While this apparently caused new epithelium formed over such areas to be more firmly adherent, blebs would occur in untreated areas which once or twice spread to include most of the central epithelium again. Finally the patient was put on fairly large doses of quinine internally and following this was free from attacks for a long time only having had one slight attack with a small bleb since the quinine was begun. She is still taking small doses and is under observation. The quinine was suggested by the older authors on account of its vaso-constricting effect.

When seen recently (January 1925) a condition greatly resembling that in Case 4 had developed. The old epithelium was full of fine vacuoles which in many areas became confluent to form large flat spaces under the epithelium, apparently containing no fluid as the level of the epithelium was not appreciably raised. In the upper part of the cornea numerous irregular folds in Descemet's were present with capacities in the deeper stroma and some pigment deposits. None of the droplets on Descemet's described by Friedenwald were present nor have any been noted in any of the other cases in this series.

I have seen several such cases following intra-ocular operations. In one man who developed glaucoma one year after cataract operation the whole corneal epithelium became loose and was peeled off. The new epithelium remains slightly wrinkled and lusterless, but is nowhere raised and the eye is no longer painful. It would seem in these cases, that either the disturbance to nutrition due to the corneal section, or the hypotension following it, produces permanent changes involving a loose attachment of the epithelium to Bowman's membrane,

or an accumulation of fluid under it. If the theory, which will be discussed later, be justified that epithelial dystrophy depends on a lesion of the corneal nerves, these cases might be explained as due to failure of the nerves to regenerate after section.

In a woman with chronic glaucoma of long standing a trephining operation done by Dr. Harold Gifford was followed by a marked disturbance of the epithelium which became so opaque that the iris was hardly visible. Later most of this epithelium peeled off and for some time showed recurrent erosions. The cornea finally became relatively clear and no more erosions occurred although the tension was still somewhat increased.

Of the classical picture described by Fuchs as Epithelial Corneal Dystrophy, I have only seen one example.

CASE 4.—Mrs. M., aged 57, stated that vision had been failing in both eyes for the past year. There had been no pain or congestion. Vision with best correction was R. E. $\frac{20}{100}$, L. E. $\frac{20}{80}$. Both corneas showed fine punctate opacities, seen especially well with a high plus in the ophthalmoscope. In the right eye there were also some fine striæ. The slit-lamp showed these striæ to be folds in Descemet's, while the punctate opacities were in the epithelium. The epithelium of the right cornea was roughened but did not stain with fluorescin, while the left cornea was covered with fine staining points and some larger areas that stained more faintly. The sensation of the right cornea was everywhere much reduced, and in some places absent entirely (on pressure with a cotton wisp). In the left cornea it was reduced but nowhere absent. Pressure with a blunt applicator showed a large area of loose epithelium below the center of the left cornea, part of which was removed with forceps. The defect had almost entirely healed the next day, which was the last time the patient was seen. Tension was R. E. 10 and L. E. 12 Schiötz. A peculiar phenomenon was noted in this patient which was also seen in case 2, a maximal dilatation of the pupil after one drop of 2% cocaine, evidence of markedly hypersensitive sympathetic nerve endings in the iris. A chronic conjunctivitis and blepharitis was present and treatment for this and for a chronic maxillary sinusitis were the only measures prescribed. Though only seen twice the clinical picture so closely resembled that of Fuchs' cases that a very chronic course with increasing loss of vision

could be predicted. The existence of hypo-tension is interesting as a probable cause of the folds in Descemet's in the right eye, where tension was slightly lower. In this eye the process was evidently of longer standing than in the left.

Differing from this picture in the age of the patients affected and in the mildness of the visual disturbance are two cases seen in the past four months. It is these mild cases which I believe can sometimes only be disposed with the aid of the slit-lamp.

CASE 5.—Mrs. M., aged 44, had been refracted in April, 1923, when vision was normal and nothing unusual was noted except a slight roughness of the lower folds and tarsal conjunctiva. A year later she appeared complaining of poor vision. Vision could not be brought above $\frac{20}{25} - 2$ in the left eye, but nothing was found to account for this. Five weeks later the patient stated that vision in the left eye was worse, varying a good deal during the day, and that it felt painful at times. Vision in the R. E. was $\frac{20}{20} - 3$, in L. E. $\frac{20}{200}$ not improved. A fine cloudiness of the cornea seen with the ophthalmoscope suggested glaucoma, but tension was found to be 13 (Schiötz). The epithelium did not stain grossly but with the slit-lamp, which was not used for the first time, a picture resembling that in the right eye of Case 2 was seen. Fine green staining dots were scattered over the whole cornea, more densely in the central part. There were many of them connected by very delicate fine grey lines, just perceptible with the highest magnification (× 80). By reflected light many of these spots were seen to be minute vesicles, evidently unruptured, and apparently no larger than one or two epithelial cells. Others had evidently ruptured, and some had become confluent to form erosions three or four times as large. The right cornea showed no staining areas, but numerous greyish opacities confined to the epithelium of about the same size as the erosions, and connected, like them, by delicate grey lines. The deeper corneal nerves could not be well seen on account of these superficial opacities. The sensation was markedly reduced over both corneas. Two days later the slit-lamp picture was about the same, but vision was $\frac{20}{40}$ in the left eye. A month later it was $\frac{20}{30}$, and no pain or congestion were present. Treatment had been confined to homatropine drops, hot applications, and an ointment for the chronic blepharitis which was present, and thought to be a possible factor in the corneal condition. Now the staining areas were less numerous, and many were replaced by greyish

spots. Some corneal nerves seen in the right eye were abnormally large. Six weeks later, when vision was $\frac{20}{15}$ in both eyes, and no congestion or pain was present, fine green staining streaks were seen in the right cornea, and many fine staining spots were still present in the left. These retained the stain $\frac{30}{60}$ minutes. Sensation was still markedly reduced in both corneas.

CASE 6.—Mr. K., aged 38, came in complaining of poor vision and frequent attacks of slight inflammation in the left eye. Vision was $\frac{20}{60}$ unimproved. The left cornea showed several small opacities just below the center, the epithelium over these being raised slightly. There was also a rough area near the upper limbus, with several small erosions. These were touched with trichloracetic acid. A chronic follicular conjunctivitis and blepharitis were present, and this was treated with a weak copper collyrium. The cornea became considerably smoother, but the lids remained much the same and after an application of 2% silver nitrate to the lids, an acute iritis developed, with very fine precipitates visible with the slit-lamp on Descemet's. This subsided on atropine and sodium salicylate, after the extraction of an infected tooth. The left cornea was now examined with the slit-lamp after an instillation of fluorescin, and a number of discrete green-staining areas were just below the center. Most of these were entirely in the epithelium, though two greyish opacities involved the superficial stroma. Grossly, no staining could be seen. A week later, five small areas were seen to retain the stain (see Fig. IV). The congestion had almost entirely disappeared, and sight was about as at first. The sensation over the whole left cornea was markedly diminished compared to that of the right cornea.

CASE 7.—Mrs. E., aged 33. Referred by Dr. Chase of Ft. Dodge, Iowa, on account of a peculiar keratitis affecting the left eye. The right eye was apparently normal. In the left eye the vision was $\frac{5}{200}$ improved with $+4.00 = +4.00$ *cx* 155 to $\frac{20}{60}$. The cornea showed four to five small opacities near the center. Sensation is absent over the whole area as tested with a fine cotton point. A history of severe attacks of inflammation in the eye with increased loss of vision during the past two years was given. The last attack had occurred three months before. Slit-lamp examination showed the opacities to be confined to the epithelium. They showed no staining with fluorescin. Dr. Chase describes the original attack as having begun with severe neuralgic pains on the left side of the face, lasting for two weeks, followed by a feeling of a foreign body in the eye.

At this time striate opacities were seen in the deeper layers of the cornea. Later vesicles were seen over these opacities which broke forming superficial erosions.

While this case was only seen once, and I have not been able to follow its further history, it resembles epithelial dystrophy of the cornea more than any other condition although it is atypical in some respects.

LITERATURE.

Since Hansen's description of intermittent keratitis neuralgica vesicularis in 1872 and Arlt's report in 1874, reports on traumatic and spontaneous recurrent erosion of the cornea have been fairly frequent, especially in the German literature. Von Szily, Senior, described his own case in 1884 and reviewed the subject completely in 1900. Other important contributions are those of Peters (1904), Francke (1906), von Szily, Junior (1913), and Salus (1922), the last giving the literature to date. The clinical picture is always much the same. In the traumatic cases, some days after a slight trauma to the eye, such as removal of a foreign body, an attack of intense pain occurs, often on opening the eyes after awakening from sleep. At this time, the epithelium is seen to be raised in a bleb of varying size, or if the bleb has broken, to be loose, and on its removal the epithelium for some distance around is found to be loose, so that often most of the corneal surface is denuded. This looseness of the epithelium around the lesion was found in all of von Szily's 15 cases, and is a constant sign. It was absent in two of his cases just after the original injury, but these later developed typical attacks, when the epithelium became loose. After removal, the epithelium is rapidly regenerated, but is only loosely attached and other attacks occur, with no further trauma than opening the lids. These may stop after a time, but usually continue until all loose epithelium is removed, and the surface touched with an astringent such as aqua chlori, when the new epithelium becomes firmly attached and no more attacks occur. The spontaneous form resembles this description except in the absence of any history of trauma. Cases are not so common as those of the traumatic form and are more obstinate in recurrence. Several of von Szily's cases were spontaneous as were Salus' four cases. Von Szily, Junior,

saw two cases after cataract operation, which were like my Case 4, and another after dislocated lens and iridectomy. He thinks these cases indicate that injury to the endothelium may be the primary cause in some cases. After ulcers, especially dendritic ulcers, as Peters points out, the same looseness of epithelium may occur. I have seen it, as Peters also reports, after disciform keratitis and in one case of smallpox infiltrate. In all these conditions, there is evidently some disturbance which allows fluid to collect between the epithelium and Bowman's membrane, whether or not the epithelium is first detached by trauma. Sections of removed epithelium by von Szily, Senior and Junior, Peters and Francke, showed the cells to be soaked with fluid, pushed apart and more or less necrotic. Franke's sections showed that fluid may collect between the epithelial cells themselves, leaving some cells attached to Bowman's membrane. The question, why the erosions recur in some cases after slight trauma and not in others, and why they occur spontaneously at all have been answered in different ways. In animals, Francke could not produce the condition by trauma alone, but by rubbing in irritants such as tuberculin and killed streptococcus cultures, the epithelium became loose and erosions recurred after two to seven days. An individual predisposition must be assumed which allows fluid to collect under the epithelium, and this is generally thought to be some disturbance of the corneal nerves analogous to that in neurotic skin diseases, where vesicles are often formed. While lesions of the endothelium may be the cause in post-operative cases, or after severe trauma, this can hardly be true of the majority of cases where the injury is insignificant, or of the spontaneous cases. It seems just as likely that the section of nerves by a cataract incision may in some cases be followed by faulty regeneration, resulting in disturbed circulation of lymph in the cornea. Peters thinks edema of the cornea, due to vasomotor disturbances, occurs, and that the excess of the fluid is forced out under the epithelium. Salus agrees with Peters, and explains the mechanism of the edema as due to the purely sensory stimulus of a trauma affecting a hypersensitive vasomotor center to produce an increased flow of lymph from the vessels at the limbus into the cornea. In the so-called spontaneous cases, he supposes a very irritable center which reacts

with edema to common slight traumata such as opening the lids after sleep. Hence these are more prone to recur than the cases occurring after greater trauma where the irritability of the center is increased, and reacts to such an irritation with edema, but is not so greatly increased as to cause further attacks when once healing is sufficiently firm. He explains the favorable effect of quinine given internally by its vasoconstrictor effect.

What Fuchs has called Dystrophy Ephithelialis Corneæ, while probably similar in pathogenesis, presents a somewhat different picture. Of this, as of so many other conditions, it is true that little of importance has been added since Fuchs' classical description. In 1910 he reported 13 cases, observed in ten years, five of which were unilateral, 5 bilateral, and three affecting eyes with increase of tension. All of his cases were above the age of 50 except one, who was 48. Vesicles occurred in most cases, but were small, and besides the vesicles, punctuate spots appeared in the epithelium and more diffuse dots and stripes were present in the superficial parenchyma, under and around the vesicles, visible when the epithelium was removed. He describes one mild case, resembling my Cases 5 and 6, in which many fine vesicles were always found in each eye for six years, but the vision remained $\frac{5}{5}$ and $\frac{5}{6}$, a slight improvement over that present at the outset. In the other cases, however, vision gradually decreased through an increase in the opacities, reaching $\frac{6}{60}$ or only ability to count fingers in those cases followed long enough. No cases were cured and no treatment seemed to be of any effect. Scraping off the epithelium and using iodine on the bare surface produced only temporary improvement, the results sooner or later recurring. In striking difference to recurrent erosion the trouble always began insidiously, with slight or no pain or irritative symptoms, the first sign being always diminution of vision which varied greatly from day to day and even hour to hour. Halos were often seen, as in glaucoma, but increase of tension only occurred in three cases. The sensation of the whole cornea was always greatly diminished and often absent. In the five monocular cases, some disturbance of sensation was noted and from this Fuchs concludes that the condition is primarily a disturbance of nutrition affecting the superficial corneal

nerves. This disturbed sensitivity was noted in all my cases, sensation usually being absent in some areas and greatly diminished in others. Fuchs made sections of one piece of tissue removed by trephine for examination from an old case. The epithelium was very irregular in thickness, and raised in places by masses of coagulum, since small vesicles were present between the layers of the epithelium itself. A layer of new connective tissue separated the epithelium from Bowman's membrane. No causal factor could be found for the condition except the age of the patients and the predominance of the female sex, nine cases being women and four men. Soon after Fuchs' report, several authors described similar cases. P. Knapp, in 1911, saw the typical picture develop after cataract operation accompanied by increased tension. He believed it to be an edema of the epithelium, as it cleared up temporarily after each massage of the cornea. Vision was reduced to counting fingers. A. Knapp, in 1911, also reported a typical case after cataract operation but also affecting the other eye. Hoppé's case (1912) was followed for ten years, the right eye being affected five years after the first, and vision gradually decreasing till the patient's death. De Schweinitz reported two cases in 1913, one coming on thirteen years after cataract operation. Motolese, Whitham, Reese, Troncoso, and Uhthoff have each reported one typical case. Clegg saw both eyes of two sisters affected with the simple condition and two other cases with glaucoma. Whitham has reported three fairly typical cases, all of whom had diabetes. These are the only cases in the literature in which the presence of diabetes is recorded. Whitham believes on account of the favorable response to dietetic treatment that this or some allied disturbance of metabolism may be a factor in causing the condition. In one of his cases slit-lamp examination by Dr. John M. Wheeler (the only record of such an examination of this condition except that of Kraupa and Vogt that I can find) showed a picture a good deal like that seen in the present cases. Troncoso's was unusual in being only 28 years of age when the condition had existed for a year. Uhthoff made sections of an excised fragment, finding a less advanced stage than that reported by Fuchs. The epithelial cells were swollen and partly fused to a homogeneous mass. The superficial stroma was

also edematous and Bowman's membrane considerably broken up. No signs of inflammation were present. Stoewer, believing that the friction of the lids played a part in keeping up the irritation, treated his case by drawing conjunctival flaps over the upper and lower parts of the cornea, after shaving off the epithelium, in the hope that this would protect the central portion. No fresh opacities or vesicles occurred during four months' observation. Aside from this case, none of the reported cases showed decided improvement on any form of treatment. Of the nineteen cases found in the literature since Fuchs' report, eleven were women, six men; nine were bilateral, five unilateral. (In several, this data was not given.) Four showed definite glaucoma, and several others slight temporary increase of tension. The ages ranged from 28 to 82, but only two were under fifty. The only American cases reported are those of de Schweinitz, Whitham, Reese, A. Knapp, and Troncoso (Mexico).

While only these cases could be found which conformed in all important particulars to Fuchs' description, several other conditions have been described which are perhaps atypical forms of the same disease, or at least may be considered as similar in nature. The "Striped Dystrophy of the Cornea" described by A. von Szily, Junior, showed very fine lines in the epithelium which stained with fluorescin, and changed in arrangement rapidly, *i.e.*, within a few hours. This condition lasted several months but cleared up without permanent opacities in one case, while it continued during his observation in another. The stripes corresponded to the corneal nerves, and were thought to be due to faulty nutrition of the epithelium adjacent to them. Believing this whole group of cases to be dependent on primary disease of the corneal nerves, he proposes a classification as follows.

1. Keratitis neuroparalytica. Lesion of main 5th nerve trunk.

2. Herpes keratitis and dendritic ulcer. Lesion of larger branches.

3. Striate epithelial dystrophy. Lesion of sub-epithelial and intra-epithelial plexuses.

4. Neurotic fine punctuate epithelial dystrophy. Lesion of finest nerve branches and end-bodies.

A somewhat similar condition was described by Holmes-Spicer as "Flame-shaped Epithelial Keratitis," one of his patients being a cook. Von Szily's first case was a baker and he thinks exposure to heat was a factor. Henderson's case of "Superficial Central Keratitis" was an engine fireman and presented a similar picture, which cleared up fairly rapidly, however.

The condition described by Staehli as "Minute Defects of the Corneal Epithelium" would apparently fall in von Szily's last class. He saw a number of cases complaining of pain and irritation in which no lesions were to be seen by ordinary methods, but which, on illumination with the Nernst light (not the slit-lamp) showed with high magnification numerous (often thousands) of minute points which stained green with fluorescin. They were chiefly in the central part of the cornea, and sometimes arranged in lines. The corneal sensation was markedly decreased. The condition was usually very chronic and resistant to treatment. This is interesting as the picture resembles closely that seen in my Cases 5 and 6 and in the right eye of Case 2, and suggests that some cases in which the ordinary examination fails to explain symptoms of poor vision and irritation would fall into this class if examined by high magnification with sufficient illumination.

Koeppe's "Keratitis Epithelialis Punctata," as studied in two cases with the slit-lamp, differed from Fuchs' picture in showing no vesicles or staining areas, in the absence of any opacities in the stroma, and in being of short duration, but probably also depends on some lesion of the corneal nerves. The "Superficial Keratitis" which Duverger and Lampert studied with the slit-lamp showed erosions and decreased corneal sensitivity, but was accompanied by a zone of opacity in the middle layers of the cornea and also by precipitates and other evidences of iritis. It was cured by tarsorrhaphy maintained for two months. In Myoshita's "Keratitis Superficialis Diffusa" the picture of multiple fine staining points in the epithelium was present as in my Cases 5 and 6. Sections, however, showed evidences of active inflammation, and the fact that all his cases occurred after acute conjunctivitis indicates that the disease is entirely different in pathogenesis from Fuchs' condition. Another difference is its acute course,

with complete healing. Cases of keratitis neuro-paralytica may present a similar picture, as in Chance's case, which first showed edema of the epithelium with fine opacities, and then developed recurrent erosions of almost the whole cornea. In these cases the pathogenesis would be similar to that of recurrent erosion, while in keratitis e lagophthalmo, a perfect picture of mild epithelial dystrophy may be present, as in one case which I studied with the slit-lamp, due purely to exposure, and disappearing completely when the facial paralysis cleared up. A similar picture is also shown in Igersheimer's illustrations of chrysarobin keratitis, but due here purely to the chemical irritant.

I have seen cases of steamy cornea in glaucoma which also showed numerous staining areas as in these mild dystrophic cases.

CONCLUSIONS.

The purpose of this paper is to call attention to the relation between Recurrent Erosion of the Cornea and Fuchs' Epithelial Dystrophy and to describe a condition which is evidently a mild or very early form of the latter disease. The relation is emphasized by Case 2, in which one eye showed this mild condition of multiple minute epithelial defects and the other typical recurrent erosions. It will be interesting to see if these mild cases go on to develop the typical picture of Fuchs. At any rate, the study of these cases with the slit-lamp has apparently given good evidence against the opinion of Kraupa, who examined later cases showing folds in Descemet's and concluded that the disease is primarily an endothelial dystrophy. In these mild or early cases, folds in Descemet's were never observed, the lesions being confined to the epithelium. Kraupa and Vogt and Whitham are apparently the only observers of the condition who have reported slit-lamp findings, and it is to be hoped that further observations will clarify the subject of these milder dystrophies. My cases have all shown a chronic blepharoconjunctivitis, but whether this was a causal factor or purely secondary is uncertain.

There seems to be no doubt that both Recurrent Erosion and Epithelial Dystrophy are dependent on lesions of the finer

corneal nerve branches or endings. Peters' theory of an edema on a vaso-motor basis seems to apply, not only to Recurrent Erosion, but also to Epithelial dystrophy, where the slit-lamp picture may greatly resemble the steamy cornea of glaucoma, though normal tension is usually present. Where glaucoma does occur, it probably is only another manifestation of vaso-motor instability.

It is suggested that the name Epithelial Dystrophy of the Cornea be applied to both these conditions and also to such allied conditions as von Szilys' "Striate Keratitis," and "Keratitis Epithelialis Punctata," the more exact condition being indicated as "Minute Epithelial Dystrophy," "Epithelial Dystrophy with Recurrent Erosions," etc.

NOTE—Since writing the above the report on H. & J. Friedenwald has been received. The fact that in one of their cases folds in Descemet's were seen at a time before epithelial changes were present is offered as evidence that the dystrophy is primarily an endothelial one. This evidence would seem to be more than offset by the early epithelial changes in my Cases 2, 5, and 6, endothelial changes being entirely absent at this stage.

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ILLUSTRATING DR. S. R. GIFFORD'S ARTICLE ON "EPITHELIAL DYSTROPHY AND RECURRENT EROSION OF THE CORNEA AS SEEN WITH THE SLIT-LAMP."

R. E. L. E.

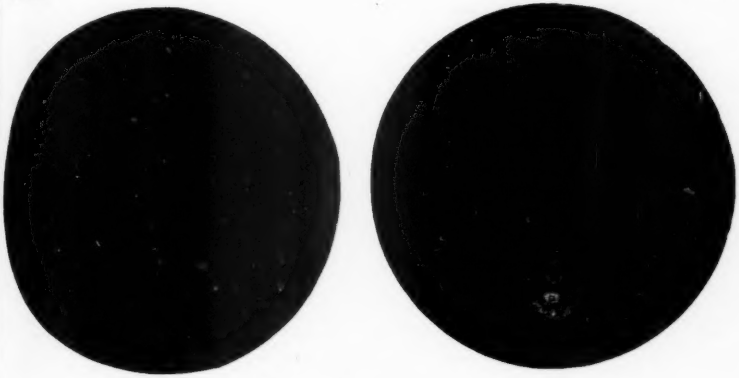

FIG. I.

Case 2, December, 1922

R. E. L. E.

FIG. II.

Case 2, September, 1924

In the left eye the central erosion has increased in size. The grayish opacities seen outside of this area may have been present at the first examination but were not noted.

ILLUSTRATING DR. S. R. GIFFORD'S ARTICLE ON "EPITHELIAL DYSTROPHY AND RECURRENT EROSION OF THE CORNEA AS SEEN WITH THE SLIT-LAMP."

R. E. L. E.

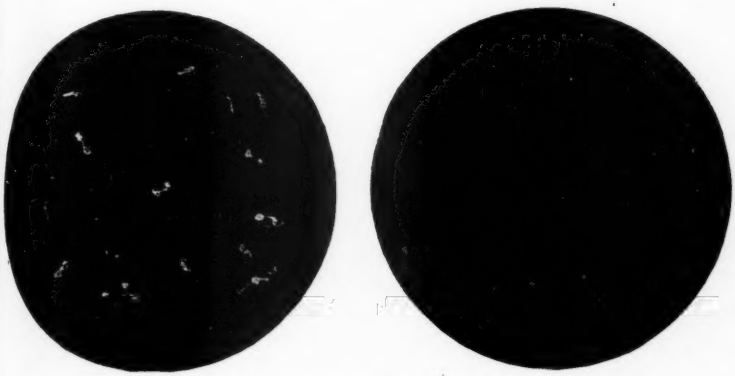

FIG. III.

The right eye shows grayish opacities which suggest a relation to the corneal nerves. The left eye shows numerous punctate erosions.

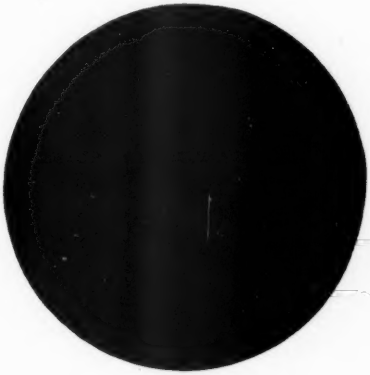

FIG. IV.

Left eye of Case 6.

PROJECTION AND DOUBLE VISION; SOME NEW VIEW-POINTS.

By Dr. ALEXANDER DUANE, New York.

(With four figures in the text.)

THE current conception of projection, both monocular and binocular, contains much that is confusing and some things that are contrary to experience. It may be well then to reconsider the subject in some of its bearings, particularly as these are by no means devoid of practical interest.

MONOCULAR PROJECTION.

In monocular projection we judge of the position of an object partly from the impression that its image makes on the retina (*visual projection*), partly from the labyrinthine and muscular sensations which indicate to us the actual direction of the eye itself (*postural projection*). Thus if we are sighting with one eye an object *A* we judge *A* to be straight ahead of us if its image falls on the fovea and at the same time the sensations derived from movements of the head and the eye assure us that neither head nor eye is turned. On the other hand, we judge *A* to be situated 20° to the right, if it forms its image on the fovea, but to get it there we have to turn the eye or the head through 20°—the sense of muscular effort exerted giving us quite an accurate notion of the movement made in following *A*. Finally we judge *A* to be situated 20° to the right, if when these same muscular impressions assure us that the eyes and head are straight, visual impressions tell us that *A* forms its image 20° to the left of the fovea.

These and like examples show that in vision with one eye, we always get our notions of the position of objects from a

combination of visual and postural projection. The former tells us that some one object, as *A*, lies in the direction in which the eye is pointing and also tells how other objects *B*, *C*, etc., are situated with regard to *A*. It does not by itself tell us how *A*, *B*, and *C* are situated with regard to us, for though it indicates that *A* lies in the direction in which the eye is pointing it does not tell us what that direction is. This information is afforded by sensations emanating from the muscles of the eye and neck and from the labyrinth, *i.e.*, by postural projection. Visual projection thus gives *objective*, postural projection *subjective* orientation.

If either element is disturbed, we project falsely. Thus, if when the eye is at rest and sighting an object *A*, a prism is interposed, the image of *A* no longer falls on the fovea and *A* itself appears displaced. Postural projection here is unaffected but visual projection has been deranged, and consequently our judgment of the position *A* is rendered faulty. On the other hand, in cases of paralysis visual projection is unaffected, but the erroneous impressions conveyed from the paralyzed muscles give a false idea of how the eye is directed and this derangement of postural projection causes us to locate falsely an object which the eye is attempting to follow.

These exceptional cases apart, the combined effort of visual and postural projection gives us a surprisingly accurate idea of the location of external objects both with regard to each other and to ourselves. Monocular projection is thus usually performed with considerable precision. With regard to it, the following facts must be noted:

1. Of the two factors, visual and postural, the latter is dominant, muscular sensations often compelling us to modify the impressions derived from our visual sensations and always in the direction of increased accuracy. On this point I shall not dwell here, having treated it at length elsewhere.

2. Postural projection in monocular vision is effected by sensations derived either from movements of the head or from purely conjugate movements of the eye, *i.e.*, such movements as the eye makes in parallelism with its fellow. In this regard it differs essentially from binocular projection.

3. These sensations give us an accurate idea of the direction in which the eye is pointed and, when coupled with vis-

ual sensations, an accurate idea of the direction of external objects, but do not, except imperfectly, give us a notion of the distance at which the objects are situated. In other words it affords orientation in space in a transverse but not in a fore-and-aft plane, telling us, for example, how much an object *B* is to the right, left, above, or below an object *A*, but not telling how much nearer or more remote it is.

LAW OF MONOCULAR PROJECTION.

As has just been said, in monocular projection the postural element and the visual element combine to give us our notions of the placing of objects. The net result of the combined action of these two elements may be summed up in the following law:

No matter how the head or the eye is turned—right, left, up, or down—and no matter how the eye is rotated in its fore-and-aft axis in performing any physiological act, postural (i.e., muscular) impressions always make the eye project as if it had not moved at all.

Thus, if we turn either the head or the eye 20° to the right to sight an object, the muscular impressions conveyed make us appreciate that although the object forms its image on the fovea it is situated just where we should judge it to be if both eye and head had kept still and the object had consequently formed its image on the retina 20° to the left of the fovea. Again, if we turn the eye up and to the right to sight a vertical line, that line appears vertical, in spite of the fact that theoretically as a result of the physiological torsion of the eye in this position it should appear tipped. It appears, in other words, as it would if the eye were looking straight ahead and the line making its impression on the lower left-hand portion of the retina was located by visual projection alone. Finally when the head is tilted to the right the vertical meridian of the eye tilts with it. Nevertheless, a vertical line at which the eye is looking remains vertical, even though theoretically, as its image sweeps over every meridian of the retina in succession, it should appear to revolve to the left. The corrective effect of muscular sensations (postural projection) is such as to make the eye see the line just as it would if

the head had not moved and the vertical meridian of the eye had remained vertical.

BINOCULAR PROJECTION.

In binocular projection there are as in monocular projection both visual and postural elements. The visual element helps us to localize an object through the impression that its retinal image produces. In this respect binocular is like monocular projection, with the important difference that in the former there are two simultaneous and usually not quite identical retinal images of the same object which reinforce each other so as to increase materially the clearness of the impression and add to it a sensation of depth.

With regard to the postural element in binocular projection there are two factors to be considered. First, as in monocular projection there are the sensations derived from the head movements and from conjugate movements of the eyes which make us aware that the latter are directed right, left, up, or down. Second, there are the sensations derived from movements of convergence. These latter sensations are the distinguishing feature of binocular projection, differentiating it from monocular projection and serving to explain all the phenomena of binocular diplopia. This factor hence requires careful consideration, which can be had perhaps best by reference to Fig. 1.

Suppose the eyes L and R are turned in by an equal converging effort to sight the object A. The left eye L, made aware of its inward turning by the muscle sense, would, if acting by itself, receive the impression that A is situated on the right and in fact somewhere along the line LA. Similarly the right eye, R, acting by itself would receive the impression that A is on the left and indeed somewhere along the line RA. But if binocular were conducted like monocular projection neither impression would tell whereabouts on LA and RA, A is. For when the left eye by the conjugate (eyes-right) movement employed in monocular vision turns toward A, the muscular effort involved is the same no matter where A is situated along the line LA. But when both eyes turn together toward A, the position is reached not by

a conjugate movement but by an effort of convergence, which is precisely equal in the two eyes. The result is that

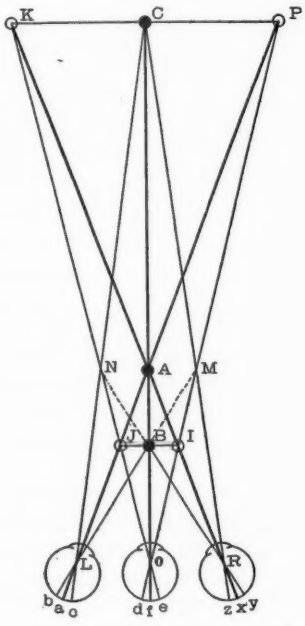

FIG. 1.

PROJECTION FROM THE BINOCULUS; BIFOVEAL SINGLE VISION AND PHYSIOLOGICAL DIPLOPIA.

L and *R*, nodal points of left and right eye. *A*, object of binocular fixation, forming its image on *a* and *x*, the foveæ of the right and the left eyes. *B*, object nearer than *A*, forming its image on the retinal points *b* and *y*. *C*, object more remote than *A*, forming its image on the points *c* and *z*. *LA*, *RA*, visual lines of the left eye and right eye, converging upon *A*. *O*, nodal point of the binoculus (binodal point) representing the fusion of *L* and *R*. *f*, the fovea of the binoculus (bifovea), representing the fusion of *a* and *x*. *d*, point in the retina of the binoculus representing the fusion of the corresponding points *b* and *z*. *e*, point representing the fusion of the corresponding points *c* and *y*. *OA*, the bivisual line, representing the fusion of *LA* and *RA*. *M*, *N*, location of the physiological double images of *B* and *C* according to the common explanation. *I*, *J*, true location of physiological (crossed) double images of *B*. *K*, *P*, true location of the physiological (homonymous) double images of *C*.

the mind receives the simultaneous impression that *A* is as much to the right of *L* as it is to the left of *R*—in other words, that it is in the mid-line *OAC*. Moreover, since the converging effort increases *pari passu* with the angle of convergence, the sensation conveyed is that *A* is nearer than an object *C* on which one would have to converge less and further than on object *B* on which one would have to converge more. Furthermore, *A* appears single because the similar, but usually not quite identical, images formed on the foveæ, *a* and *x*, of *L* and *R* are combined by the process we call fusion into a single, composite image affording more or less the impression of depth and relief.

The net result then of the two distinctive factors of binocular projection, viz., fusion, or the visual element, and convergence, or the postural element, is that *A* or any other point which is the object of bifoveal fixation is

(a) Considered always to lie in the mid-line *OAC*.

(b) It is regarded as at a more or less definite point on that line and, in fact, in about its true place.

(c) It appears single.

Quite the same is true if *A* while forming its image on the fovea in each eye is not situated straight ahead but to one side, say the right. The only difference is that to the equal converging effort which each eye is making and which tells us that *A* is in the mid-line between the eyes is added a conjugate movement which tells us that, as both eyes while fixing *A* are swung to the right, *A* itself is also situated on the right.

We may in fact conceive binocular projection, as performed by a single cyclopean eye, or to use Maddox's useful phrase, a *binoculus* (*O*) situated in the mid-line and with its visual line directed along *OAC*. The fovea of this eye (*f*) representing the combined foveæ, *a* and *x*, may be called the *bifovea; O*, representing the combined nodal points *L* and *R*, may be called the *binodal point;* and the visual line *OAC*, representing the fusion of the two visual lines *LA* and *RA*, may be called the *bivisual line*. The bivisual line may be directed straight ahead, or sideways, or up and down, but its direction is always given by a line connecting the binodal point *O* with the object of fixation *A*.

PROJECTION BY THE BINOCULUS; BINOCULAR SINGLE VISION.

Projection by the binoculus is performed just as it would be by each eye, if each were transferred to the root of the nose and then rotated, the left eye to the left and the right eye to the right until the fovea of each was in the mid-line (*a* and *x* transferred to *f*, and *LA* and *RA* made to coincide with *OA*). Any object whose image falls simultaneously on the two foveæ *a* and *x*, or as we may say, on the bifovea, *f*, is projected along *OA*.

The binoculus may be regarded as a concept by which we can best denote the result of the fusion of binocular visual impressions in the cerebral cortex and the way in which these fused impressions are re-projected out into the external world. It represents the fusion not only of the right and left foveæ and of the right and left visual lines, but also the fusion of each pair of corresponding points in the two eyes. Thus not only does *f* represent the fusion of *a* and *x*, but *d* represents the fusion of *b* and *z*, and *e*, the fusion of *c* and *y*. The points *b* and *z* being linked together by their connections in the left cerebral cortex, convey ordinarily a sense of identity of position and are hence projected out to the same point in space. It is exactly as if instead of projection being made from the two points *s* and *t* of *L* and *R*, respectively (Fig. 2), it is made from the point *u* bearing the same relation[1] to the bifovea, *f*, that *s* does to *m*, and *t* does to *n*. *u* in this case is the peripheral representative of the cortical impression produced by the fusion of the two sensory impressions sent up from *s* and *t*.

With the binoculus thus we gain binocular single vision and also the correct placing of external objects, when the

[1] If *u* bears exactly the same relation to *f* that *s* does to *m* and *t* to *n*, and if projection is made from *u* strictly on the basis of sensory perceptions, then the combined visual impressions set up by a body *D* would be reprojected out, not to *D* but to a point *E* a little nearer the mid-line. Apparently jected out, not to *D* but to a point *E* a little nearer the mid-line. Apparently, however, no such false projection takes place, the sensory impressions here as generally being corrected by the muscular impressions, so that *D* is not seen at *E* but in its true place.

latter form images simultaneously in the two foveæ or simultaneously on any pair of corresponding points.

These facts, which are of fundamental importance, may perhaps be put better in another way. Since the binoculus

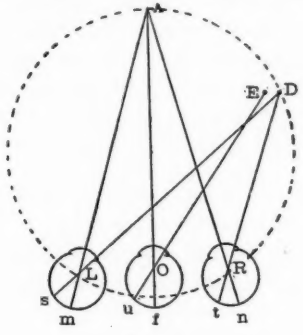

FIG. 2.

PROJECTION BY THE BINOCULUS; CORRESPONDING POINTS.

L and *R*, nodal points; *m* and *n*, foveæ of the left and right eyes, respectively. *s* and *t*, corresponding points, lying equally far and in the same direction from the fovea in the two eyes. *LA*, *RA*, visual axes. *O*, binodal point; *f*, bifovea; *OA*, bivisual line; *u*, point in the retina of the binoculus, representing the fusion of *s* and *t*. *A*, object of binocular fixation. *D*, an object forming its image on the corresponding points *s* and *t*. *E*, point to which *D* would be projected from *u* according to the rules of simple visual projection. *D*, however, owing to the corrective effect of muscular sensations, is probably as a rule projected out to its own place. In any event *D* appears single. *D*, it will be observed, lies in the horopteric circle (dotted line) passing through *A*, *L*, and *R*. Compare with this figure Figure 1, in which the corresponding points [1] *b* and *z* would project out to the one point *M* the images falling on them.

is simply the representative of the linked visual centers in the cerebral cortex and is therefore a representative of the combined retinæ of the right and left eye, every point in the retina of the binoculus projects like the corresponding point in the retina of each component eye, but the projection is

[1] This explanation is not affected by the fact that the horopter probably has not the form of a circle as given in Fig. 2. The statement remains true that in case any point *D*, whether in the horopteric circle of Müller or not, does form its image in the corresponding points *s* and *t* it will be projected by the binoculus as if from *u*.

made with reference to the mid-line (bivisual axis) instead of with reference to the right or left visual axis as it is in monocular vision. This means that when the left eye, *L* (Fig. 1), is acting by itself, stimulation of a point *b* 15° to the left of the fovea will be regarded as set up by an object 15° to the right of the visual line *LA:* but when *L* is acting in conjunction with *R* (and under the influence of convergence), stimulation of *b* will be regarded as set up by an object 15° to the right of *OA*. So too, when *L* is acting alone stimulation of *a* will mean for it an object situated somewhere along *LA*, but when *L* is acting with *R*, stimulation of *a* will mean an object situated in the mid-line *OA*, and in fact at its intersection with *LA*—*i.e.*, at *A*.

PROJECTION BY THE BINOCULUS AS AN EXPLANATION OF PHYSIOLOGICAL DIPLOPIA.

It remains to consider how projection with the binoculus takes place when the retinal images of an object fall neither on the two foveæ nor on any extrafoveal pair of corresponding points.

Let us first regard the physiological diplopia observed when we look sharply at an object *A* (Fig. 1) and at the same time take notice of objects *B* and *C* respectively nearer and further than *A*. Both *B* and *C* appear double, the images in the former case being crossed, in the latter case being homonymous.

The explanation commonly given for this fact that, when the eyes are fixing *A*, *B* appears in crossed double images, is that the left eye projects *B* out to *M*, and the right eye projects it out to *N* in the plane of *A*. But the most simple experiment serves to show that this is not true. If we hold up a pencil a foot from the eye and regard a wall one or two yards away, the pencil splits into two. The double images, however, simply separate laterally, so that the pencil appears no further off after division than it did before. There is no appearance suggesting projection on the remoter wall. If there were such a projection, we should lose to a certain extent our notion of the distance of the wall, since the pencil images apparently coinciding with it would no longer appear less remote. But as a matter of fact, the two pencil

images appear not on the wall but as far away from it as ever, so that they still give as great an idea of the relative distance as before, and, moreover, the separation of the images gives an added clue to this, since the greater this separation is, the further away from the pencil we judge the wall to be.

The error in the current explanation lies in the fact that it is based on the assumption that in binocular vision each eye projects as it would if seeing monocularly. If each did so, then there is really no reason why *B* should not appear single, since the left eye projects it along *LB* and the right eye along *RB*, and as it is not projected beyond its own place, the two images should lie at the point where *LB* and *RB* cross—*i.e.*, should be coincident.

The true explanation must fit the fact that when we look at *A*, a nearer object *B* doubles and yet looks no further off than before. The following explanation which is essentially that given years ago by Le Conte[1] may serve to show that projection by the binoculus affords a ready means of accounting for this.

When the two eyes are fixing *A*, the images of *A* on the two foveæ *a* and *x*, are fused in the cerebral cortex and as we have seen, are projected by the binoculus as from the bifovea *f*, straight out along the mid-line *OA*. *A*, moreover, appears in about its true situation. The left eye image of *B*, *i.e.*, *b*, 15° to the left of *a*, produces an impression in the cerebral cortex which imparts the sensation of an object say 15° to the right of *A*. It is thus by the binoculus projected as from *d* 15° to the left of *f* out along the line *OI*. Since it is judged to be no further off than *B*, it appears to lie at *I*. In a similar way, the right-eye image of *B* is projected out from *y* 15° to the right of *f*, along the line *OJ*, and is judged to lie at *J*. *B*, therefore, appears in double images *I* and *J*, *I* the left-eye image being on the right and vice versa.

By a reasoning entirely analogous, it will be clear that *C*, more remote than *A*, will appear in homonymous double images, *K* and *P*, the former corresponding to the left-eye image, *c*, the latter to the right-eye image, *z*, and both lying in the plane of *C*.

---

[1] *Sight.*

PROJECTION BY THE BINOCULUS AS EXPLAINING PATHOLOGICAL DIPLOPIA.

Quite like the explanation of physiological diplopia is that of the diplopia occasioned by deviations of the eyes or by prisms. In both cases the dominance of convergence sensations makes each eye project as if from the mid-line binoculus.

To understand this suppose we consider the case of esotropia. The essential element in this is a primary or secondary excess of convergence which makes both eyes tend to converge nearer than the point of fixation. Since rarely, if ever, is a convergence effort unequally distributed between the two eyes, both eyes in this case though converging excessively converge to the same extent. Hence instead of being directed at the point of fixation *C* (Fig. 3) both are turned in equally to the nearer point *A*. In that case just as shown in Fig. 1, *C* will appear in homonymous double images, *K* and *P*, on either side of *C*. But *A* will not appear clear since the eyes are not accommodated for it but for *C*, and the visual desire to get a clear image of *C*, will compel one of the eyes to move in such a way as to get this image on the fovea. Suppose it is the left eye that does this. To do it it will have to swing to the left until it points directly at *C* and the image of the latter hence falls on the fovea *a*. This leftward swing is a conjugate movement shared equally by the right eye. Hence when the left eye has swung to the left through the angle *ALC*, the right has swung also to the left through an equal angle. The two eyes therefore now have the positions shown in Fig. 4, the left eye pointing toward *C*, the right deviating from the latter twice as far as before and pointing toward *G*.

As the left eye swings to the left the projected image *K* of the object *C* swings to the right until it finally gets to occupy *C* and appears then clear and sharp. At the same time, the projected image *P* of the right eye swings equally to the right and occupies the position *H* (Fig. 4). As it does this it becomes more indistinct since its retinal counterpart now is twice as far removed from the fovea as before.

It will be observed that the squinting angle, which was *KOP* when the eyes were both converged on *A* and which is now *CRG*, has remained unchanged in this process of rota-

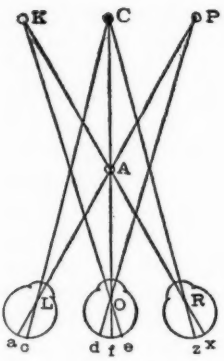

FIG. 3.

DIPLOPIA IN ESOTROPIA.

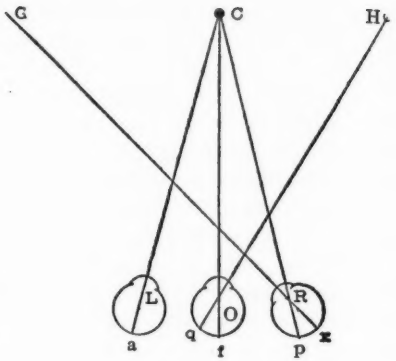

FIG. 4.

DIPLOPIA IN ESOTROPIA; SUPPRESSION; PERVERSE DIPLOPIA.

Fig. 3. Both eyes are converged equally as in the case shown in Fig. 1, but, owing to the excess of converging power, both are directed at an object *A* nearer than the object of fixation, *C*. *C* is thus seen in homonymous double images, *K* and *P*. Notation same as in Figure 1, to which reference is made for explanation of diplopia.

Fig. 4. Same case as in Fig. 3, but both eyes have now swung to the left, so that the left eye is pointing at *C*. As it receives the image of *C* on the fovea *a*, it projects this image as if from *f*, *i.e.* in the bivisual line and out to *C*. The right eye is turned in twice as far as before, namely to *G*, 30° to the left of *C*. It thus receives the image of *C* on the point *p* 30° to the left of the fovea *x*. But, in spite of this deviation, the right eye continues to follow the law of binocular projection because it is still under the domination of the sensations set up by convergence, which, as shown in Figure 1, is equal for the two eyes. It hence like the left eye projects as if its visual line, *RG*, were coincident with the bivisual line, *OC*. Consequently the object *C*, which forms its image on *p*, is projected as from the corresponding point, *q*, of the binoculus, *i.e.* out to *H*. If the right eye should happen to lose completely its association with the left and escape entirely from the domination of convergence impulses, it would project simply according to the laws of monocular vision. That is it would not project the image falling on *C* out to *H*, 30° to the right of the bivisual line *OC*, but would project it 30° to the right of its own visual line, *i.e.* out to *C*. In this case there would be no diplopia, but a conflict of superposed images leading to suppression. If the right eye has not escaped wholly from the domination of convergence and is in part only governed by the impressions afforded by conjugate impulses, which rule monocular vision, the image on *p* will be projected somewhere between *C* and *H*; *i.e.* the diplopia will still be present, but will be less than the deviation (perverse diplopia).

tion. It will also be observed that the double images produced, viz., *C* and *H*, are in character and in origin essentially like those produced in physiological diplopia.

Thus the images *C* and *H* in Fig. 4 differ from the images *K* and *P* in Fig. 1 only in the fact that the former have both been shifted over to the right by an amount equal to half the distance between them. Moreover, like *K* and *P*, *C* and *H* are produced by projection from the binoculus, *C*, which represents the image formed on the fovea *a* in the left eye, being projected as from *f* in the bivisual line *OC*, and *H*, which represents the image formed on *p* in the right eye, being projected as from the corresponding point *v* of the binoculus. Thus in the case of an ocular deviation, as in the case of binocular fixation, all images formed on the retina of either eye are projected as if both eyes were united in the binoculus and their visual axes coincided with the bivisual line. And this is so because in the one case as in the other the two eyes are subjected to an equal converging effort. Such an equal converging effort forms, as we have seen, the postural basis of mid-line projection.

The only real difference between the conditions represented in Fig. 4 and that shown in Fig. 1 is that in the former the diplopia affects the object of fixation, while in the true physiological diplopia, shown in Fig. 1, it does not. In the latter case there are seen a pair of shadowy double images accompanying a third, single and distinct, image which represents the object of fixation; while in the pathological diplopia there are two images, one shadowy, the other distinct, of the object of fixation itself.

Quite analogous is the explanation of the crossed diplopia produced by a divergence of the eyes.

Paretic diplopia finds essentially the same explanation. As long as the impulses arising from equal convergence are dominant, the object fixed by either eye will be regarded as lying in the bivisual line, and an object whose image lies at some outlying point like *p* (Fig. 4) will be projected from the corresponding point *q* of the binoculus, *i.e.*, will appear to bear to the bivisual line *OC* the same relation that it actually bears to the monocular visual line. In other words, the effect on the projection of the equal converging impulses sent to

both eyes is so strong that each visual axis *LC* and *RG* is still conceived of as occupying the mid-line position *OC*, no matter how either axis may actually be dislocated. And it makes no difference in this regard to what cause this dislocation of the axis may be due, whether excessive or faulty convergence, excessive or faulty divergence, or paretic weakness of one of the muscles. The convergence impulse in all these cases remains the same, equal in the two eyes and controlling the projection.

We need not dwell on vertical diplopia, to which a similar reasoning applies, nor on diplopia produced by prisms. The latter case is obviously the same as that of a pathological deflection of the eyes. In both cases diplopia is due to the fact that an image which to produce single vision should fall on the fovea, actually falls on some extrafoveal point, and the only difference is that in a deviation of the eyes the fovea is deflected away from the image, in the diplopia produced by prisms the image is deflected away from the fovea.

RÔLE OF CONVERGENCE IN BINOCULAR PROJECTION.

From the above it is apparent that just as conjugate movements,—*i.e.*, movements of one eye in parallelism with its fellow—are the essential feature in furnishing postural projection in monocular vision, so convergence movements are the distinctive element of postural projection in binocular vision. It is the sense of convergence effort, emanating equally from the two eyes, and impelling both toward the mid-line, that gives necessarily the impression that the object looked at by both eyes is in the mid-line and that it is at a certain distance. This convergence effort therefore effects two things. First, it transfers the two visual axes to the mid-line and makes us project accordingly; second, it enables us to locate objects in a fore-and-aft plane.

We are also able to locate objects in a transverse plane since in addition to the movements of convergence there are side-to-side and up-and-down movements (conjugate movements of the eyes or head) and the muscular efforts involved in these afford a notion of how the two eyes are thereby directed. The amount by which the eyes are thus turned in response to conjugate movement is indicated by

the direction of the bivisual line, or line extending from the binodal point to the object of fixation.

The overmastering influence of the convergence impulse makes itself felt even when the two eyes do not converge on the same object, so that there is diplopia. In fact, diplopia can hardly be explained otherwise. For, as we have seen, an eye, even when deviating, habitually projects as though postural—*i.e.*, convergence—impressions made it locate its visual axis in the bivisual line and made it refer its visual impressions to that line, just as the non-deviating eye does. Thus in Fig. 4, the right eye projects as though its visual axis *RG* were coincident with *OC*, in this regard, acting just like the non-deviating left eye. Thus we can still speak of a bivisual axis even though there is but one object of fixation. We may say, indeed, that binocular vision is governed by convergence posture as monocular vision is governed by conjugate posture.

PERVERSE DIPLOPIA AND SUPPRESSION.

There are certain cases, however, in which a deviating eye escapes more or less from the influence of the postural influence of convergence and even in binocular vision acts, in some regards at least, as though governed by the impressions set up by conjugate movement, *i.e.*, by the impressions that dominate monocular sight. Such a partial reversion to a monocular state explains, I believe, the conditions denoted as suppression and as perverse or paradoxical diplopia.

To make this clear we have only to take such a case as that shown in Fig. 4 and inquire what would happen if the deviating eye *R* projected the object *C*, as it would do, if the other eye were shut and *R* remained pointing toward *G*. In that case *R* would project the object *C*, not with reference to the bivisual line, *OC*, but with reference to its own visual axis *RG*. By impressions conveyed by the muscles acting on it and effecting a conjugate movement, *R* would become aware that it was directed to the left and in fact actually pointing toward *G*. *G* would appear in its proper place and *C* would be projected to the right of *A* and through the angle *RAC*,—therefore also to its proper place. If *R* projected thus when both

eyes were open, there would appear two coincident images of *C*, one seen by the right eye, the other by the left. The condition, however, would not be the same as in ordinary binocular single vision, for while the left-eye image of *C* would be distinct, the right-eye image, being located on an extrafoveal part of the retina, would be indistinct. Whenever such images of the same object are superposed the distinct image tends to extinguish the other, until it finally disappears altogether. This would be the case here no matter whether the actual deviation of *R* was greater or less. No matter how this deviation varied, *R* by getting from monocular—*i.e.* conjugate—muscular sensations a notion of its true direction would project the object fixed by the other eye to its proper place, and when thus projecting it would lose sight of it (suppression).

Such a suggestion would seem to afford at least a plausible explanation of the suppression that takes place in the ordinary strabismus of childhood.

Now suppose that the reversion to the monocular type of vision (projection by conjugate instead of convergent impulses) was not complete as in the case just pictured but partial and more or less varying. In that event the deviating eye *R* would become not perfectly but only approximately aware of its true direction, so that it would get the impression that it was pointing more or less toward *G* but not actually at it. Its visual axis then would neither be regarded as having its true direction *RG* nor yet as being transferred to *OC*, but as having some intermediate direction. In that case, *C* would neither be projected to its true place as when the reversion to the monocular type of projection was complete, nor to *H*, as when the binocular impulse was entirely dominant, but to some point intermediate between *C* and *H*. There would thus be a diplopia, but less than the amount that would correspond to the deviation. Moreover, while the deviation remained the same, the amount of the diplopia might vary, depending on which set of impulses, convergent or conjugate, got the upper hand.

This would explain, I think, satisfactorily the conditions found in perverse diplopia, in which the images are less far apart than they should be and the amount of the double vision

changes often considerably while the deviation remains the same.

This viewpoint may be summed up in the statement that the difference between ordinary diplopia, suppression, and perverse diplopia lies in the fact that in the first case the deviating eye, dominated by convergence impressions, locates its visual axis in the mid-line *OC;* in the second case, being wholly under the influence of conjugate impressions, it locates its visual axis where it actually is, *i.e.*, in *RG;* and in the third case, being under the influence of both convergence and conjugate impulses, it locates its visual axis along some intermediate line. *C* is projected according to its relation with the visual line as thus located.

There is no doubt that monocular projection (projection set up by conjugate movements) is of earlier development than binocular projection, which is set up by convergence action. Perverse diplopia and suppression in which monocular projection more or less prevails over binocular represents then a reversion to the primitive type.

SUMMARY.

1. Monocular projection is partly the result of visual impressions (visual projection), partly the result of sensations emanating from the labyrinth and from the muscles of the head and the eyes (postural projection).

2. Of the two factors in projection, the postural element is predominant correcting the impressions received from the visual element, always in the interest of accuracy.

3. Visual projection affords objective orientation telling us how objects are situated with regard to each other; postural projection affords subjective orientation telling how objects are situated with regard to the beholder.

4. Since the postural sensations in muscular vision are derived from conjugate movements only, they effect orientation in a transverse, but not in a fore-and-aft plane. That is, monocular projection tells us how far one object is to the right, left, above, or below another, but not how far it is behind or in front of it.

5. In monocular projection, no matter how the head or

eye is moved and no matter how the eye is rotated on its antero-posterior axis, postural sensations always make it project as if it had not moved at all.

6. In binocular projection there is also a visual and a postural element. The visual element is that of monocular vision, but since there are two retinal images which reinforce each other the resulting impression is more distinct and acquires a stereoscopic quality.

7. The postural element in binocular projection is twofold, consisting of sensations derived from conjugate movements, as in monocular projection, and of sensations derived from convergence. The latter are the distinctive and predominating feature of binocular projection.

8. The element derived from convergence makes the object of binocular fixation appear in the mid-line. It also makes it appear at a definite distance from the eyes and thus effects orientation in a fore-and-aft plane.

9. Binocular projection may be conceived as taking place from a composite cyclopean eye or binoculus (Maddox). In the binoculus the converging visual axes of the right and left eye are fused into a single axis (bivisual line) directed in the mid-line of the head; the nodal points of the two eyes are fused into a single binodal point on the bivisual line; and the two foveæ are fused into a single fovea (bifovea).

10. Projection by the binoculus is a projection such as would be performed by each eye if each were transferred to the root of the nose and then rotated, the left eye to the left and the right eye to the right, until the visual axis, the fovea, and the nodal point of each coincided with the mid-line. By this arrangement not only are the two foveæ fused into one bifovea, but each pair of corresponding points in the two retinæ is represented by one point in the retina of the binoculus, which bears the same relation to the bifovea that the corresponding points in the right and left eye do to the foveæ of the latter.

11. Accordingly, an object which forms its image in the fovea of either eye is projected as if from the bifovea of the binoculus, and any object whose image falls on a point distant any number of degrees from the fovea in either eye is projected as if from a point having a like relation to the

bifovea. That is, an object which is fixed by either eye will appear to lie in the bivisual line; and an object whose image lies 20° to the left of the fovea in either eye will appear to lie 20° to the right of the bivisual line.

12. This principle serves to explain satisfactorily not only bifoveal vision, but also explains single vision in the case of objects whose images occupy corresponding points, and physiological double vision.

13. In most cases of ocular deviation, this same law of postural projection derived from convergence sensation prevails, and owing to this the diplopia corresponds in amount and kind to the deviation.

14. In other cases of ocular deviation, it seems probable that projection from convergence sensations is partly or wholly replaced by projection from sensations derived from conjugate movement—*i.e.*, sensations of the sort that characterize monocular projection. If the replacement is partial, a diplopia less than that correspondent to the deviation is produced (perverse diplopia). If the replacement of convergence by conjugate projection is complete, then there is no diplopia, but a superposition of the two images formed by the two eyes. In this case one of the images, being faint, is readily and soon habitually suppressed. It is thought that this may explain the suppression that takes place in squint. In other words, the replacement in squint of binocular by monocular vision would be due to the predominance of the monocular type of postural projection, and to the elimination of the sensations derived from convergence that are the guiding force in true binocular vision.

INTRADERMAL TEST WITH UVEAL PIGMENT IN TRAUMATIC IRIDO-CYCLITIS.[1]

By Dr. ARNOLD KNAPP, New York.

ALAN WOODS[2] has published studies on the immune reactions following injuries to the uveal tract in which he found that there developed in the blood serum in these cases a complement fixation reaction to an antigen made from the pigment of the uveal tract. This reaction was positive when normal healing in the first eye took place without disturbance in the fellow eye; it was absent when healing was delayed in the injured eye and sympathetic disturbance in the second eye sometimes occurred. In one case of sympathetic disturbance this complement fixation reaction was not only absent but there appeared a definite hypersensitiveness to pigment. This author also believed that the occurrence of the complement fixation was evidence of the development of an immunity by the organism to the pigment and gave protection against the outbreak of a sympathetic disturbance in the fellow eye. The failure to develop this reaction indicates failure to develop immunity and is attended by a persistence of inflammatory symptoms and by the liability to the development of a sympathetic disturbance in the fellow eye. The possible clinical value of this test if the premises are correct is evident; the development of an immunity would therefore warrant the continuation of conserva-

[1] Read before American Ophthalmological Society, Hot Springs, June 18, 1924.

[2] Woods, A. C. "Immune reactions following injuries to the uveal tract." *J. A. M. A.*, 1921, 77, 1317.

tive treatment in a case of injury while contrariwise the outbreak of sympathetic disease is a factor to be reckoned with.

Dr. Woods (personal communication) now thinks "that the technical difficulties, constant and exact titration of the antigen for each separate set of tests, instability of the antigen, close titration required, and large number of controls needed, all tend to make the complement fixation test of little practical value, outside of a research laboratory where it is in constant use."

"The intradermal test should theoretically give more pertinent and precise information as to the possibility of an impending sympathetic ophthalmia. It is quite easy to do, requires no especial apparatus, and the pigment when preserved in sealed ampules seems to hold up well for several weeks at least. For the above two reasons it seems that the complement fixation reaction will be of little use, while the intradermal test should be of a great deal of use, provided that the early promise of the test is substantiated."

The technic of the intradermal test is as follows: Three different dilutions of the normal pigment suspension (the pigment of one cow's uveal tract to 7.5 *cc* of salt solution is called "normal") are used. The solutions are 1:50, 1:10, and normal (full strength). Using a fine hypodermic needle, small intradermal injections, sufficient to raise a bleb about one-half the diameter of a dime, are made with each dilution on the flexor surface of the fore-arm. A control is made with 0.15 per cent. tricresol in normal salt solution. A positive reaction is shown by the development of an erythematous area sometimes with a central bleb about the pigment suspension injections. This comes on within an hour and lasts for a number of hours. In a negative reaction no redness or swelling develops.

This intradermal test was tried out in twelve cases of traumatic irido-cyclitis during the past winter at the Herman Knapp Memorial Eye Hospital with uveal pigment which Dr. Woods had been kind enough to send me. The results were as follows: In four the test was positive; in two of these, sympathetic irido-cyclitis was present in the second eye; in two, irido-cyclitis did not develop in the second eye. The test was negative in eight: in one, sympathetic irido-cyclitis

developed in the other eye, in seven, the second eye remained normal.

The cases in brief are as follows:

CASE NO. 1.—J. S. Aged 48. Traumatic irido-cyclitis. Pigment test positive.

Injured by a piece of wood on February 6th, 1923. There was a perforating wound of the left cornea with incarceration of iris and capsule, traumatic cataract and irido-cyclitis. On March 27th, 1923, the ciliary congestion was still present and there was an exudate at the corneal wound. Pigment test suspicious. There was a negative reaction about control, $\frac{1}{50}$ and $\frac{1}{10}$ dilutions, but full strength or normal suspension showed erythema of the size of a 25 cent piece coming on within 45 minutes and only beginning to fade about two hours after injection.

Two months later, June 1st, the eye had continued red and there were many posterior corneal deposits. Second pigment test: Positive. Considerable erythema (size of a 50 cent piece) about all injections within 15 minutes, but control soon faded out. The other persisted for two hours and then gradually faded, the strongest disappearing last.

June 14th, 1923. As eye has remained red though projection and tension were normal, enucleation was done.

Microscopic examination: Several groups of cells composed of lymphocytes and plasma cells are situated in the iris and ciliary processes. The choroid is not involved. There is a wound of the lens covered by organized cellular exudate and in the lens there is an area composed of broken-down pus cells and endothelial cells, a picture which Dr. Verhoeff, who was kind enough to examine the specimen, regarded as typical phaco-anaphylactic endophthalmitis. The iris angle contained some lens debris and a few epithelioid cells.

CASE NO. 2.—A. W. Aged 10. Sympathetic irido-cyclitis. Pigment test positive.

Admitted to Hospital November 1st, 1923. About one year ago shot in face and eyes by discharge from shot-gun. Left eye—phthisis bulbi: cystic condition of iris; organized vitreous exudate, retracted iris. Right eye: white, anterior chamber shallow: almost total posterior synechia: iris atrophic. Organized pupillary exudate. Fundus reflex, no details. V. $\frac{20}{200}$; T. 31.

November 2nd, 1923. Pigment test—positive. Erythema about all three pigment injections, but none with the control. Erythema $1\frac{1}{2}$ inches about normal suspension:

faded out from $\frac{1}{10}$ (weakest) in 45 minutes, but persisted about the normal suspension about 3 hours.

November 3rd, 1923. Enucleation left eye.

Microscopic examination: Intense infiltration of iris, ciliary processes, and choroid with the typical cell changes found in sympathetic inflammation: lymphocytes, epithelioid cells, and giant cells. Organized cyclitic membrane within the vitreous, retina detached. Optic neuritis.

CASE No. 3.—J. F. Aged 4. Sympathetic irido-cyclitis. Pigment test positive.

Admitted to Hospital December 13th, 1922. History: Right eye struck by buckle on October 10th, 1922. November 8th, 1922, left eye became red and the right eye was immediately removed.

December 13th, 1922. Pupil dilated. Iris atrophic. Vessels on iris. No corneal deposits. Vitreous hazy, especially in periphery just back of lens in ciliary region. Nerve head gray, blurred: and venous congestion.

December 19th, 1922. Pupil contracting. Nodules appearing in iris and more vessels visible. Pigment test positive. Erythema 1½ inches about normal suspension. Erythema ½ inch about 1:10 suspension; erythema slight about 1:50 suspension. Control—negative. The erythema began to fade in about one hour and disappeared in two hours.

Microscopic examination: Enormous infiltration of ciliary processes with lymphocytes and epithelioid cells, giant cells, breaking through at the pars plana, with proliferation of pigment and infiltration of circumlental space. Iris and choroid involved.

CASE No. 4.—D. C. Aged 42. Traumatic irido-cyclitis. Pigment test negative.

On March 8th, 1923, end of wire struck right eye causing a linear perforating wound of cornea and iris. Traumatic cataract.

March 15th, 1923. Increased ciliary congestion. Yellowish reflex from vitreous.

March 27th, 1923. Pigment test: no reaction.

Under treatment the eye would improve for a time and then again become red and painful.

April 27th, 1923. Ciliary congestion. Iris fair. Tension normal. Projection normal.

May 20th, 1923. Ciliary congestion; iris tissue is more infiltrated. Tension and projection good. Second pigment test—negative.

June 2nd, 1923. Discharged. Slight ciliary congestion. Anterior chamber deep down and out, where iris is also

atrophic. The eye gradually quieted down and has given no further trouble.

CASE No. 5.—H. T. B. Aged 53. Left eye: (old) prolapsed iris. Optic neuritis. Right eye: irido-cyclitis. Optic neuritis. Pigment test negative.

Admitted to Hospital May 28th, 1923. Two months ago a perforating injury of cornea and prolapse of iris occurred in the left eye after being struck by a stick. The prolapse was not treated. Two and a half weeks ago the sight of the right eye became affected. V. right $\frac{2}{4}\frac{0}{0}-$; left $\frac{2}{2}\frac{0}{0}\frac{0}{0}$. Left: faint ciliary congestion. Subconjunctival prolapse of iris. Much blurring and some swelling of disk. Right: faint ciliary congestion. Many corneal deposits. Distinct blurring of nerve head.

June 1st, 1923. Pigment test. No reaction.

Discharged: June 25th, 1923: V. right $\frac{2}{4}\frac{0}{0}$; left $\frac{2}{8}\frac{0}{0}$. Right—few deposits and slight blurring of disk. Left: disk still distinctly blurred. Gradual recovery. August 20th, 1923: Right $\frac{2}{4}\frac{0}{0}$; left $\frac{2}{8}\frac{0}{0}$. Right, few corneal deposits. Optic nerve normal. Left, prolapse flat. Optic nerve normal.

CASE No. 6.—N. I. Aged 50. Intraocular foreign body. Traumatic cataract and irido-cyclitis right. Pigment test negative.

On December 1st, 1922, the right eye was struck by a foreign body while chiseling stone. An intraocular foreign body was extracted with the giant magnet on December 6th, 1922. Irido-cyclitis then developed.

March 10th, 1923. Enucleation. Pigment test negative. Left eye normal.

CASE No. 7.—F. C. Aged 36. Irido-cyclitis and secondary glaucoma right; anophthalmos left. Pigment test negative.

The left eye was enucleated after an injury three years ago. Admitted to hospital August 13th, 1923, with irido-cyclitis and secondary glaucoma in only eye of two weeks' duration. Uveal pigment test negative.

Irido-cyclitis cleared up after nose, throat, and teeth were attended to.

CASE No. 8.—N. E. Aged 37. Perforating wound of cornea and sclera of left eye. Incarceration iris: prolapse of vitreous. Pigment test negative.

On February 16th, 1924, was struck in the left eye by a sharp piece of metal. There was a perforating wound extending horizontally from below center of cornea into

sclera for several *mm* with laceration of iris and prolapse of vitreous. The wound was cleansed, edges approximated and covered by conjunctival flap.

Patient slowly recovered, moderate reaction. After four weeks the congestion was localized about the wound and there was an anterior synechia of lower pillar of iris.

Three weeks after injury, pigment test was negative.

CASE NO. 9.—E. E. Aged 52. Intraocular foreign body. Traumatic irido-cyclitis. Vitreous abscess right. Pigment test positive.

Admitted *June 13th, 1923*, giving an indefinite history of injury two days before. Right: chemosis lids and conjunctiva. Annular infiltrate of cornea. *3mm* hypopyon and exudate in pupil. Very faint fundus reflex. Tension −. Right V. − H.M. X-ray negative. Under treatment (milk, anti-diphtheritic sera, etc.), the cornea and anterior chamber became much improved.

June 27th, 1923. Corneal wound nearly healed. Hypopyon absorbed. Pupil dilated. Yellowish reflex from vitreous (abscess). Tension −. Vision: Light perception but projection faulty.

July 5th, 1923. Increase in yellowish vitreous reflex.

July 17th, 1923. A second X-ray showed intraocular foreign body near ciliary body.

July 19th, 1923. Pigment test: Erythema about all pigment injections, measuring 1½ inches at the normal suspension which faded out in about 2 hours.

July 21st, 1923. Enucleation right.

Microscopic examination: Anterior chamber contains fibrin, lymphocytes, and pus cells; there is a purulent exudate on the anterior surface of iris. The iris shows moderate infiltration with lymphocytes, pus cells, and plasma cells, especially along its posterior surface. The ciliary processes are infiltrated and a cyclitic membrane with many plasma cells stretches across the vitreous with abscess formation arising from the pars plana retinæ. The choroid has distended vessels but only few cells. The retina is inflamed with purulent changes on its inner side.

CASE NO. 10.—E. P. Aged 23. Sympathetic irido-cyclitis left. Anophthalmos right. Pigment test negative.

February 20th, 1924. Struck right eye against counter which did not give any trouble until four weeks later when eye became red. He then consulted Dr. J. G. Dwyer to whom I am indebted for the case and who found a penetrating injury of the cornea with prolapse of iris though

fairly good vision ($\frac{7}{10}$). One week later the left eye became red and the right eye was enucleated.

March 20th, 1924. Pigment test negative.

No cause can be found for the irido-cyclitis in the second eye as all tests were negative. There is a gelatinous exudate in the anterior chamber. No corneal deposits. The iris is irregularly bulging with complete pupillary synechia. Tension very soft. V.: L. P.

April 5th, 1924. Second pigment test negative. Immediate erythema around all injections (pigment and control) but they all quickly faded out.

The cyclitis in the left eye has steadily advanced. There are many posterior corneal deposits. The iris is converted into nodular masses and the pupil is completely bound down. The eye is very soft and there is bare L. P.: it is undoubtedly a severe sympathetic irido-cyclitis.

The microscopic examination of the enucleated right eye shows a subconjunctival prolapse of iris at the corneo-scleral junction. This prolapsed knuckle of iris is intensely infiltrated with lymphocytes and epithelioid cells. The rest of the iris is uniformly infiltrated, the ciliary body moderately so but the choroid is most affected and progressively toward the posterior half of the eyeball. The infiltration is diffuse, is composed of characteristic cell structures, and extends into the sclera along the vessel and nerve paths. The optic nerve is slightly affected while the retina and the other structures in the eyeball are normal.

CASE NO. 11.—J. H. Aged 71. Perforating wound of cornea with prolapse of iris. Traumatic cataract and irido-cyclitis. Pigment test negative.

The right eye was struck by a piece of wood on February 15th, 1922. The prolapsed iris was abscised. Wound healed; long convalescence due to persistent irido-cyclitis.

Readmitted January 23rd, 1923, for extraction of cataract. Scar in cornea *2mm* from limbus with anterior adhesion iris. Lower part of iris is atrophic. Many posterior synechiæ and thickening of capsule. Combined extraction January 24th, 1923, followed by considerable reaction and recurrence of cyclitis.

Pigment test, March 25th, 1923, negative.

May 1st, 1923, as eye had continued red and painful it was enucleated.

Microscopic examination: Deep anterior chamber contains pus cells, lymphocytes, and fibrin. Retraction periphery of iris, ciliary body drawn inward, purulent exudate, posterior to lens capsule about ciliary processes, especially to one side. Beginning detachment retina. Exudate in anterior

vitreous is partly pus, partly organized tissue. In anterior periphery of choroid some lymphocytes and plasma cells, otherwise ciliary body and choroid are not involved.

CASE NO. 12.—E. S. K. Aged 46. Penetrating injury cornea and ciliary body. Pigment test negative.

May 7th, 1924. Struck in left eye causing a wound of cornea extending into sclera with prolapse of iris and vitreous. Wound cleansed, edges brought together and covered with conjunctival flap. Moderate reaction but no severe irido-cyclitis.

Pigment test, May 29th, 1924, negative. Gradual recovery.

Of the four positive pigment tests in two the clinical manifestations of the irido-cyclitis in the second eye and the histological changes found on microscopic examination of the injured eyes were characteristic of a sympathetic inflammation. In the remaining two where the tests were positive no inflammation developed in the second eye, the microscopic changes in the injured eyes which were enucleated did not show the evidences of a sympathetic inflammation. There were present partly a purulent infiltration and partly the reaction to a chronic infection characterized by a moderate infiltration of lymphocytes and plasma cells, localized to the iris and ciliary processes while the choroid was perfectly free. No epithelioid cells were present except for a few occurring in the iris angle of Case 1 where they seemed to be a reaction caused by lens debris.

The early histological changes in sympathetic iridocyclitis are not well-defined though the picture of the advanced disease is quite characteristic.

At the same time the authorities agree that nearly always all three parts of the uvea are involved, and most definite and greatest is the involvement of the choroid either in nodes or later diffuse with characteristic involvement of the sclera. The way of extension in the uvea is not known except that it is not continuous. In the early cases the inflammatory changes in the anterior half of the eyeball are slight and the choroid shows numerous sharply defined perivascular foci of leucocytes.

Professor E. Fuchs and Dr. F. H. Verhoeff who were kind enough to look over these specimens confirmed the presence of any evidences of sympathetic irido-cyclitis.

Of the eight negative tests, the irido-cyclitis which developed in one of these cases ran a malignant course and now presents all the clinical signs of a sympathetic irido-cyclitis. The histological examination of the injured eye gave characteristic changes. The failure of the test in this case is particularly significant and instructive.[1] In three other cases in this group an examination of the injured eye was made in two, and the changes of a mild purulent irido-cyclitis were found present.

The length of time that pigment hypersensitiveness exists is of interest. In the two cases clinically and microscopically sympathetic in nature the hypersensitiveness to pigment was present in one, A. W., one year after the injury. From the clinical appearance of the sympathizing eye where the process had come to a standstill the irido-cyclitis must have been of from six to nine months' standing. It is of interest to note that though clinically the process had come to a standstill the pigment sensitivity still remained. In the second case, J. F., of undoubted sympathetic irido-cyclitis the injury occurred on September 20th, 1922, the second eye became involved November 8th, 1922. The dermal tests made December 25th, 1922, proved positive; the eye then showing active sympathetic changes. In the third case, J. S., the injury was sustained on February 6th, 1923. On March 27th, the first skin test was suspicious. On June 1st, the test was repeated and was then found definitely positive. The cyclitis continued unabated until June 14th, when the eye was enucleated. The other eye at no time showed any evidence of irido-cyclitis and has remained well. The fourth case, E. E., received an injury to his eye on June 11th, 1923. The pigment test on July 19th was positive, the eye presenting the changes of irido-cyclitis with a vitreous abscess. The eye was enucleated on July 21st. The other eye has remained unaffected.

While the outcome of these tests for pigment sensitivity did not follow in all cases our clinical and histological evidence, the subject warrants further study.

[1] Fearing lest the negative skin tests in this case might be due to the pigment being too old, a fresh supply was obtained from Dr. Woods and another test was made on Sept. 10th, 1924, with a definitely negative result.

ILLUSTRATING DR. OTTO LANDMAN'S ARTICLE ON, "A CASE OF TUBERCULAR IRIDO-CYCLITIS WITH FATTY DEGENERATION AND INFILTRATION OF THE ANTERIOR SEGMENT OF THE EYE."

No. 1.—Fat stained red with Sudan III showing the homogeneous mass filling the angle of he anterior chamber, fatty changes in the cornea; in the exudate of the bulla; in Bowman's and Descemet's membranes; in the iris; noting the meagre fatty changes around the tubercles of the iris; fatty changes in the ciliary body, in the lamina vitrea choroidea, and in the sclera.

No. 2.—Fat stained red. The angle of the anterior chamber completely obliterated by he organized exudate which has undergone fatty degeneration; the fat is arranged radially atty degeneration of the iris. The ciliary body with tubercles and areas of fatty degeneration atty changes in cornea, Bowman's and Descemet's membranes, and sclera.

A CASE OF TUBERCULAR IRIDO-CYCLITIS WITH FATTY DEGENERATION AND INFILTRATION OF THE ANTERIOR SEGMENT OF THE EYE.

By Dr. OTTO LANDMAN, Toledo, Ohio.

(With two illustrations in color on Text-Plate X.)

HANNSEN[1] reports the fat findings in seventy human eyes, some of which were diseased and some normal. A review of his examinations discloses the fact that fat occurs frequently but not constantly in eyes which were *not* diseased, for instance, in the sclera, except in the newly born or in early youth; in the ciliary body and cornea; the lamina vitrea choroidea very early shows a red stain with Sudan, likewise Descemet's membrane, with intact endothelium shows a red stain.

This article concerns a case whose history is briefly as follows:

September 22, 1922. Mrs. Z., age 55. Her right eye became red and painful on September 5, 1922. Status Præsens: Left eye normal. Right eye, V = $\frac{1}{10}$. Irido-cyclitis with numerous posterior synechia; vitreous was hazy. Physical examination revealed a positive von Pirquet. Two months after the beginning of the disease a small nodule appeared on the pupillary margin of the iris. February, 1923. Three nodules were observed in the lower half of the iris. From now on the lower part of the cornea grew opaque, so that in July, 1923, the opacity prevented a view of the iris except in its upper part. Moreover the opaque cornea which was grey at first, became more yellow day by day, till it had an intense yellow color, the lower anterior part of the sclera adjacent to the limbus corneæ, also had a yellowish tinge. In August, 1923, a superficial vascular keratitis appeared and increased in

[1] *Klin. Monat. f. A.*, 1923, Vol. 70, p. 732.

intensity. The yellow color now covered the lower $\frac{3}{5}$ of the cornea. These conditions continued for months with no pronounced exacerbations. In June, 1924, there was secondary glaucoma and a bulla formed in the lower nasal part of the cornea, but it healed within two weeks. The treatment with salicylates, mercurials and old tuberculin was without avail. In August, 1924, the anterior part of the eye looked very yellow, even when viewed from a distance. At this time the patient finally consented to an enucleation.

The enucleated eye was hardened in a neutral formalin solution. After hardening, the eye was cut into a posterior and anterior half; the anterior half was then treated by the technic of Graff.[1]

Hardened in formalin.
Infiltrated with gelatine.
Blocked.
Sectioned on freezing microtome.
Stained in Sudan III.
Counterstained in Bohmer's Alum-hæmotoxylin.
Mounted in glycerine.

Staining on the slide was found more satisfactory. After blocking, the anterior half was bisected vertically through the cornea and sclera, and two narrow strips each four millimetres in thickness were cut out parallel to the line of bisection. Each strip was then cut into halves, frozen, sectioned and stained.

Microscopical Anatomy:

The iris, ciliary body and anterior portion of the choroid are the seats of the tubercular processes, whilst the fatty changes are found in the cornea, Bowman's and Descemet's membranes, in the exudates of the anterior chamber and in the iris, in the ciliary body, in the sclera, and in the lamina vitrea choroidea.

The fatty involvements only will be considered and are of two kinds, fatty infiltration and fatty degeneration. The most frequent is the fatty infiltration which is arranged in columns as if following paths of least resistance, as in the lamellar tissue of the cornea and sclera and in the fibrin

[1] *K. M. f. A.*, 1918, Vol. 2, p. 556.

formation in the angle of the anterior chamber. In the loose structures like the iris, irregular conglomerations of the fat cells and globules occur. The fatty degeneration is found in cells and tissue fibres.

Cornea:

The lamellæ of the cornea are in a state of fatty degeneration and many fibrillæ looked like strings of minute corals. Many of the corneal clefts are bordered by these strings of fat globules. Where the fatty degeneration of the corneal lamellæ is most marked the corneal cells were missing. In some of the cornea cells the protoplasm has disintegrated into fatty globules, but the fatty degeneration of the corneal cells is relatively rare; moreover the chromatin and protoplasm of the corneal cells are very granular. In some parts of the cornea its entire thickness is involved in the fatty changes. Descemet's membrane is deeply stained, also Bowman's, which in spots is split up into red blocks. In the cornea the exudate which had filled the bulla showed spots of fatty degeneration. There is also a marked vascular keratitis mostly around the limbus corneæ.

Iris:

The iris is infiltrated with fat masses, fat cells and globules, the fat masses seem to be coalesced fat globules. The stroma of the anterior surface is the part mostly involved, although the fat is distributed liberally throughout the iris. There is fatty degeneration of some stroma cells, and fat in the protoplasm has caused a chromatolysis, although this is not a common occurrence. There is also a radial arrangement of the fat in the iris. There is not much fat found around the tubercular deposits. The fibrinous mass, described below, in some places blends with the iris tissue so closely that it seems a part of a hypertrophied iris. The entire anterior chamber is filled with a mass made up of a homogeneous part and an organized fibrinous part. The angle of the anterior chamber in the lower three fifths of its circumference is obliterated by the organized fibrinous exudate containing minute blood vessels. Large areas of this exudate have undergone fatty degeneration. Likewise the mesodermal cells in its midst are phagocytic, their protoplasm filled with fat globules and in some instances the chromatin

is destroyed. Not every portion of the organized fibrinous mass has undergone these fatty changes. The angle of the anterior chamber which is *not* filled with the organized mass is filled with a homogeneous mass stained *faintly* by the Sudan. The mass is probably a coagulum of fibrin. In it are found very many minute globules of fat, stained red, and polymorphonuclear leucocytes in a state of phagocytosis also lymphocytes in a state of phagocytosis.

Fat occurs in the ciliary body in quantities relatively less than in the iris.

The sclera is infiltrated with fat and some of its fibers have undergone fatty degeneration. The fatty changes have affected the sclera as far as the equator; sometimes the whole thickness of the sclera is deeply stained by the Sudan. In places the sclera is infiltrated with round cells. Wherever the conglomerate fat masses are found, they give the impression of inspissated fat.

The lamina vitrea choroidea is deeply stained with the Sudan. No cholesterol crystals are found in the eye. Von Szily, in *K. M. f. A.*, 1923, has described a condition, of two eye balls, which he calls Xanthomatosis bulbi, characterized by an orange yellow color in the anterior segment of the eyes. He found large masses of fat in the vitreous and anterior chambers, fat in the ciliary body and iris and some in the inner layers of the sclera, with the cornea *practically* free from fat. There were masses of crystals of cholesterol in the iris and sclera. Although the specimen, which forms the subject of this paper, had a strikingly yellow color of the cornea and lower part of the sclera, adjacent to the cornea, and although there are no crystals of cholesterol and no fat cells in the vitreous of my specimen, still there is a similarity between Von Szily's cases and my case. In my specimen the cornea is extensively involved, not so in Von Szily's. His specimen had fat in the vitreous, and cholesterol crystals in the iris and sclera; mine had not; otherwise they are alike. All of these eyes had a secondary glaucoma. I take this opportunity of acknowledging the assistance, in the preparation of the sections, of Prof. G. Carl Huber, director of the Anatomical Department of the University of Michigan, where the work was done.

STUDIES ON THE PROTEIN CONTENT OF AQUEOUS HUMOR.

The Effects of Miotics and Mydriatics.

By Dr. Francis H. Adler and E. M. Landis.
(From Department of Physiology, University of Pennsylvania.)

Introduction.

In 1918 Wessely (1) published a paper on the effects of miotics on the normal eye. By dropping a 1% solution of eserine into a rabbit's eye, the tension of which was being recorded by a suitable manometer he found that after 10–15 minutes the pressure began to rise and reached a maximum 15 minutes later. This rise began shortly after the pupil had become miotic. It slowly sank from its high level and reached normal in ½ to 1 hour.

Wessely observed that eserine constricted the blood vessels of the conjunctiva, but gave a marked hyperæmia of the vessels of the ciliary body and iris. He also showed that after eserine the protein content of the aqueous was increased, and the amount of fluorescine in the aqueous humor after intravenous injection was markedly augmented. Further he found that by injecting calcium chloride into rabbits subcutaneously, and then instilling eserine into one eye the increase in protein in the aqueous no longer occurred. Rosenow (2) in 1915 had also found that by giving parenteral injections of 5% calcium chloride he could markedly delay the appearance of the fluorescine in the anterior chamber.

From these facts Wessely concluded that one action of eserine was to dilate the blood vessels of the iris and ciliary

body, causing an increased permeability of these vessels to protein and to fluorescine, and increasing the intraocular pressure.

It is common knowledge that in animals, the withdrawal of aqueous humor leads to a marked increase in the protein content of the aqueous of second formation. Wessely has always attributed this increased protein content to the dilatation of the blood vessels which lowering the intraocular pressure brings about; and he has shown (3) that if he keeps the blood vessels constricted by adrenalin or faradization of the sympathetic the aqueous of second formation contains a protein percentage approximating the normal.

These facts all indicate that the increase in protein in aqueous humor, however produced, is due to dilatation of blood vessels, with a consequent increase in permeability. This is in accord with Krogh's (4) recent work on the permeability of capillaries in which he makes the statement that whenever capillaries are dilated their permeability is increased.

The appearance of protein in aqueous humor after instillation of eserine, has been viewed from an entirely different point of view by Seidel (5) and his conclusions drawn from the experimental increase in protein have such far-reaching significance as to warrant a careful scrutiny of the facts.

It is Seidel's contention that an increase in the protein percentage of aqueous humor after eserine is evidence that the ciliary body has been caused to secrete. He agrees entirely with Wessely that after empyting of the anterior chamber the protein increase is due to the hyperæmia produced (6), but in a later paper (5) he attributes the increased protein after instillation of eserine to increased "secretion" by the ciliary epithelium.

This point is the premise on which his further reasoning depends. Since the ciliary epithelium is caused to secrete by eserine—a drug which is known to cause most gland cells to secrete—he reasons that the ciliary body is a true gland, and aqueous humor a true product of secretion.

What evidence is there that an increase in protein content can be used as a criterion for glandular activity? It would seem logical to attribute the action of eserine in increasing the protein content of aqueous to some effect on the blood

vessels, as Wessely had done. The only proof that this is not the case is Seidel's statement that he could find no apparent change in the blood vessels after pilocarpine which gave a slight increase in the protein content. After atropine instillation Seidel found the blood vessels dilated but no protein increase.

Since 1918 Seidel has contributed some twenty papers on the source and course of the intraocular fluids, supporting the hypothesis of the secretory nature of the ciliary body and of the aqueous humor. The most striking piece of evidence which Seidel produced we believe to be in the paper referred to above. If we grant Seidel the premise that protein appearing in the aqueous after pilocarpine and eserine is due to stimulation of the ciliary epithelium, then it is tantamount to a proof of the secretory nature of the ciliary body. It is more than academic, therefore, to inquire into the evidence supporting this premise. It becomes a matter of real importance, for it is needless to more than point out that our knowledge of the pathogenesis and our methods of treatment of glaucoma must ultimately be based on a correct idea of the physiological processes normally concerned. We therefore set out to repeat Seidel's work and to devise other experiments which would throw light on this problem.

The normal aqueous humor contains approximately 0.025% protein. Possible explanations of an increase in the protein under the influence of eserine or pilocarpine are:—

1. Eserine dilates the intraocular blood vessels, and thereby makes them more permeable to the protein molecules.

2. The powerful contraction of the iris under the miotics leads to a stretching of the iris blood vessels, with a resultant increased permeability.

3. The miotics have no effect on the permeability of blood vessels but actually stimulate the ciliary body to secrete, and we must suppose, with such violence that protein is turned out in the aqueous.

Methods Used.

In order to repeat Seidel's work we began our studies with the method he employed. The determination of the protein

content was made by reading the refractive index of the aqueous with the Abbe refractometer. We used Seidel's table of the equivalent protein percentage determined by him.

This method is far too inexact for such small changes as are obtained, and a glance at Seidel's table, a part of which is given below, will serve to show how easily one might be led astray in the variations occurring normally. Even the Pulitzer-Zeiss refractometer, which is about ten times more accurate than the Abbe, is too inexact for the determination of the changes as they occur in a solution of protein of approximately 0.025%.

TABLE I.

SHOWING PROTEIN PERCENTAGES IN AQUEOUS HUMOR OF CATS WITH EQUIVALENT REFRACTIVE INDEX.

Seidel—Graefe Arch. f. Ophth. Vol. 95, 1918, p. 8.

1.	n = 1.3353	0.026	
2.	n = 1.33544	0.025	0.025%
3.	n = 1.3352	0.025	
4.	n = 1.3355	0.043	
5.	n = 1.3356	0.04	
6.	n = 1.3357	0.0526	
7.	n = 1.33578	0.0526	0.058%
8.	n = 1.3357	0.07	
9.	n = 1.3358	0.07–0.08	
10.	n = 1.3358	0.086	
11.	n = 1.33589	0.073	0.084%
12.	n = 1.3358	0.12	

Wessely (7) has ably criticized the use of this method although it has been commonly employed. The variations in the experimental results lie almost within the range of experimental error. For the sake of comparison, however, we give a table of our results obtained by this method.

EXPERIMENTS.

I. *The Effect of Eserine on the Protein Content of Aqueous.*

A. Refractometer Method.

Five cats were used. 1 drop of 1% solution of eserine was instilled into one eye at ½ hr. intervals for two drops. Half an hour later the animal was etherized by open drop method, taking precautions to shield both eyes from the ether vapor, and 0.6*cc* of aqueous withdrawn from each eye by means of Luer syringes with fine needles. Table (2) gives the refractive indices and the corresponding protein percentages.

TABLE II.

SHOWING THE CHANGES IN REFRACTIVE INDEX, AND THE EQUIVALENT PERCENTAGE OF PROTEIN IN AQUEOUS HUMOR AFTER THE ESERINE.

DATE EXP. NO.	REFRACTIVE INDICES AT 17.5° C.		CORRESPONDING PROTEIN PERCENTAGES	
	Normal Eye	*Eserine Eye*	*Normal Eye*	*Eserine Eye*
(1) 1-11-24	1.33530	1.33546	0.025%	0.030%
(2) 1-12-24	1.33506	1.33518	[1]0.025%	[1]0.025%
(3) 1-12-24	1.33490	1.33496	[1]0.025%	[1]0.025%
(4) 1-15-24	1.33550	1.33665	0.040%	0.6%
(5) 1-17-24	1.33527	1.33548	[1]0.025%	0.030%

In Exp. 4 only is there a definite enough change in the refractive index to assert with confidence an increase in the protein percentage.

In order to show how the values for the individual readings vary, the following protocol is presented of Exp. No. 4.

Protocol of Exp. No. 4—1-15-24

Cat. 1 drop 2% eserine right eye at 4.50 P.M.
1 drop 2% eserine right eye at 5.20 P.M.
Anæsthesia at 6.25 and samples withdrawn from both eyes.

[1] The actual figures are slightly below 0.025%, but could not be read on Seidel's graph as the curve in this region is almost a straight line.

Normal Eye

Refractive index at 17.5° C.

Readings by F. H. A.	*Readings by E. L.*
1.3353	1.3355
1.3356	1.3356
1.3355	1.3355
1.3354	1.3355
1.3356	1.3355
1.33548	1.33552

Average of two sets of readings = 1.33550 = 0.040%

Eserine Eye

Refractive index at 17.5° C.

Readings by F. H. A.	*Readings by E. L.*
1.3364	1.3368
1.3365	1.3367
1.3368	1.3368
1.3366	1.3366
1.3366	1.3367
1.33658	1.33672

Average of two sets of readings = 1.33665 = 0.6%

Notice that readings on the same fluid vary as much as 2 or 3 points in the fourth decimal place, which according to Seidel's table in this range will give a change of 100% protein. This is sufficient proof we feel that the refractometer method is wholly unreliable for estimations of protein in aqueous humor.

In Wessely's (7) hands a precipitation of the protein was accomplished by Esbach's fluid, using very small tubes, and comparing with a set of standards. We found this method also unsatisfactory. The precipitate came down in varying physical states, sometimes very fine aggregates and occasionally quite heavy, which made readings very difficult. The

precipitate in the standards tended to adhere to the sides of the tubes which made them change. Temperature was a factor which also had to be controlled. In the region of protein percentages with which we were working, one could scarcely distinguish a 50% difference. We therefore abandoned this and at the suggestion of Dr. D. Wright Wilson adopted the nephelometer method described by Dennis and Ayer (8) for the determination of protein in cerebrospinal fluid. Using this method with some additional refinements we cut down our experimental error to approximately 2%.

B. Nephelometer Method.

The aqueous was withdrawn by means of fine platinum needles on Luer syringes, withdrawing the same amount from each eye at a uniform rate. Great care must be exercised in the withdrawal of the aqueous; it must be withdrawn immediately without damage to the iris. Any damage to the iris or delay in the withdrawal of the fluid will cause a transudation of blood serum from the vessels of the iris with consequent increase in the protein content.

A standard protein solution was made up of human blood serum, the nitrogen in the mixture being determined by the Kjeldahl method. This strong standard was preserved with thymol, and kept in the refrigerator. From this standard every two weeks a dilute standard was made up to contain 0.02% protein.

1*cc* of distilled water was added to each of two nephelometer tubes and to one of these was added 0.4*cc* of the dilute standard, and to the other 0.4*cc* of aqueous humor. 1*cc* of a 5% solution of sulphosalicylic acid was then added to each of the tubes; the mixture was very gently shaken and allowed to stand exactly five minutes. 5*cc* of distilled water was then added to each tube, and the contents of each tube poured into a separate glass beaker, rotated gently to secure even mixing and poured back into the nephelometer tube.

The tubes were then read in a Bausch and Lomb nephelometer. The standard and unknown solutions were measured out in fine glass pipettes calibrated by mercury to contain 0.4*cc*. The pipettes were emptied into the distilled water

in the nephelometer tubes and washed out thoroughly three times. Each tube was discarded after one use. All glassware was washed three times through tap water, distilled water, alcohol, and ether in succession, and dried by compressed air. The nephelometer and light were kept in a constant position on a table for all readings. Five readings were taken on each sample and averaged. All the readings were made by one of us (F. H. A.) to insure a constant personal equation.

Table 3 shows the results of eight experiments in which 2 drops of a 1% solution of eserine salicylate were instilled in one eye at ½ hour intervals, and the aqueous withdrawn from both eyes ½ hour later.

TABLE III.

SHOWING CHANGE OF PROTEIN CONTENT OF AQUEOUS AFTER INSTILLATIONS OF ESERINE.

Date	PROTEIN PERCENTAGES		*Percentage Change*
	Normal Eye	*Eserine Eye*	
2–9–24	0.024%	0.043%	+79%
2–9–24	0.023	0.036	+56
2–9–24	0.020	0.026	+30
2–11–24	0.030	0.043	+43
2–12–24	0.023	0.030	+30
5–7–24	0.017	0.020	+18
6–7–24	0.040	0.048	+20
6–25–24	0.024	0.043	+79

Average increase = 44%

II. *The Effect of Atropine on the Protein Content of Aqueous Humor.*

Table (4) gives the results of seven experiments in which two drops of a 2% solution of atropine sulfate at ½ hour intervals were instilled in one eye, and after waiting ½ hour the aqueous was withdrawn from both eyes simultaneously.

The table shows that in three cases there is an increase in the protein after atropine, and in the other cases a decrease. We find no marked constant effect of atropine.

TABLE IV.

SHOWING THE EFFECT OF ATROPINE ON PROTEIN CONTENT OF AQUEOUS HUMOR.

Date	PROTEIN PERCENTAGE IN		*Percentage Change*
	Normal Eye	*Atropine Eye*	
2-14-24	0.0266%	0.0318%	+19%
2-14-24	0.0246	0.0236	—4.0
2-15-24	0.0286	0.0278	—3.0
2-16-24	0.0555	0.0434	—2.0
2-26-24	0.0161	0.0166	+3.0
2-26-24	0.0421	0.0354	—13
2-26-24	0.0207	0.0247	+19

Average percentage change = +1.8%

III. *The Effect of Eserine on the Protein Content of Aqueous After a Previous Atropinization of the Eye.*

Seidel reported that after atropine the effect of eserine was not so marked. We therefore instilled 2 drops of 2% atropine sulphate in one eye ½ hour apart and ½ hour later 2 drops of 1% eserine salicylate ½ hour apart. The animal was etherised ½ hour after the last drop and the aqueous from both eyes withdrawn. Table V gives the results of these experiments on four cats.

TABLE V.

Date	PERCENTAGE PROTEIN		*Percentage Change*
	Normal Eye	*Atropine and Eserine Eye*	
2-12-24	0.041%	0.020%	—51.6
2-12-24	0.0195	0.0180	— 7.6
2-14-24	0.0293	0.0259	—11.0
2-16-24	0.0145	0.0152	+ 4.8

Average per cent change = —16.3

In three cases the protein percentage is lower in the eye with atropine and eserine than in the normal eye. In one

case it is slightly higher, but nowhere comparable to the effect of eserine alone. (See Table III.) This shows conclusively that atropine can prevent the increase in protein which eserine normally produces.

Criticism of Experiments.

Thus far we have confirmed the findings of Seidel as follows:

1. Eserine produces an increase in the protein content of aqueous humor.

2. Atropine gives variations in the protein content sometimes above, but more often below the normal.

3. Atropine prevents the increase due to eserine. Neither eserine nor atropine caused any precipitate with the reagent used for precipitating the protein, nor any change in the physical state of the protein in aqueous humor, which caused it to precipitate more readily than normal aqueous.

We further determined the differences in protein content of the two normal eyes in the same animal.

IV. *The Normal Variations in the Protein Content of the Two Eyes.*

TABLE VI.

SHOWING THE DIFFERENCES IN PROTEIN BETWEEN THE TWO EYES OF NORMAL CATS.

Date	*Right Eye*	*Left Eye*	*Difference*
2–23–24	0.0278%	0.0274%	1.5%
3–11–24	0.0192	0.0189	1.5
12–17–24	0.0387	0.0389	0.5
12–17–24	0.0317	0.0314	0.9
12–18–24	0.0303	0.0314	3.6
12–19–24	0.0377	0.0429	13.7

Average per cent change = −3.8%

The figures show that there is little difference in the protein content of the two eyes of a normal animal. The last experiment, showing a difference of 13.7% was due to the fact that some difficulty was experienced in withdrawing the aqueous from the left eye. If this figure is left out the average difference between the two eyes is 1.6%. This represents the prob-

able difference in two normal eyes, and is in the range of the experimental error of the method used. We included the last figure because it represents the maximum error in the rest of the experiments, and all changes under experimental conditions must be compared with this figure.

V. *The Influence of Pilocarpine on the Protein Content of the Aqueous.*

Table (7) shows the effect of two drops of a 2% solution of pilocarpine muriate given at ½ hour intervals, and withdrawing the aqueous from both eyes ½ hour later.

TABLE VII.

Date	PROTEIN PERCENTAGE		*Percentage Change*
	Normal Eye	*Pilocarpine Eye*	
6-9-24	0.2800%	0.3801%	+3.4
6-9-24	0.0332	0.0341	+2.6
6-20-24	0.0391	0.0362	—7.6
6-23-24	0.0555	0.0602	—8.4
6-23-24	0.0488	0.0472	—3.2
6-27-24	0.0311	0.0307	—1.2

Average per cent change = —2.4%

We are unable to confirm Seidel's results of an increase in protein due to pilocarpine.

VI. *The Effect of Extirpation of the Ciliary Ganglion.*

If Seidel's contention is correct that eserine increases the protein content by stimulating the ciliary epithelium to secrete, it should be possible to prevent this action by destroying the 3rd nerve fibers innervating the ciliary epithelium, since eserine produces its effect by stimulating the peripheral nerve endings. On the other hand if the action of eserine is on the blood vessels of the uveal tract then destroying the nerve endings would not prevent this effect of eserine.

In order to destroy the 3rd nerve endings we extirpated the ciliary ganglion on one side in a series of cats and allowed sufficient time for degeneration of the peripheral fibres to occur. The removal of the ciliary ganglion in cats has been described by H. K. Anderson (9) and we followed his opera-

tive technique for which reference should be made to his article. In our first few cats we waited only one week after operation before completing the experiment, but we found that the protein content was increased in the denervated eye due to the trauma of the operation itself. It was necessary, therefore, to wait one month for complete recovery before the animal could be used.

Table 8 shows the effect on the protein content of aqueous of extirpating the ciliary ganglion in four cats. In three of the cats there was a significant decrease in the protein content on the denervated side for which we have no satisfactory explanation.

TABLE VIII.

SHOWING THE EFFECTS ON THE PROTEIN CONTENT OF EXTIRPATING THE CILIARY GANGLION.

Date	PROTEIN % *Normal Eye*	PROTEIN % *Denervated Eye*	*Percentage*
4-30-24	0.0200%	0.0130%	—35 %
5-7-24	0.0293	0.0297	+4.3
5-16-24	0.0558	0.0240	—57
6-6-24	0.0403	0.0301	—25

Average per cent change = —28.2%

In another series of five cats similarly operated on the effects of eserine were tested on the denervated eye. The results are given in Table 9.

TABLE 9.

Date	PROTEIN % *Normal Eye*	PROTEIN % *Denervated and Eserine Eye*	*Percentage Change*
5-5-24	0.0175%	0.0139%	—20%
5-7-24	0.0344	0.0325	—5.5
6-6-24	0.0298	0.0325	+8.3
5-28-24	0.0209	0.0262	+25.0
5-28-24	0.0313	0.0361	+15.0

Average per cent change = +11.4%

This table shows an average increase in the protein content of only 11.4% as contrasted with a 44% increase due to eserine acting on a normal eye. The removal of the ciliary ganglion, however, lowers the protein content on an average 28.2% so that this figure must be added to the average result brought about by eserine acting on a deganglionaed eye. We then obtain a figure of +39.6%, closely approximating the effect of eserine on the normal eye. The removal of the ciliary ganglion therefore, has not prevented eserine from increasing the protein content. We conclude from this that the effect of eserine is not due to any action on the ciliary epithelium as a gland.

Table 9 gives a summary of the average effects obtained in these experiments.

Discussion of Results:

Eserine produces a definite increase in the protein content of aqueous which atropine successfully prevents when given prior to the eserine. Extirpation of the ciliary ganglion, on the other hand, is not effective in preventing the action of eserine. This would seem a paradox and as yet we have no satisfactory explanation. One of the effects of atropine is a paralysis of 3rd nerve endings. It can not be this effect which checks the action of eserine, since the mechanical destruction of these fibres by ciliary ganglion removal fails to do so. We are forced to the hypothesis that atropine has an additional effect on the eye besides its paralyzing action on the 3rd nerve endings. The most plausible explanation would be a direct action on blood vessels. This action can be either a vaso-constrictor one or by some unknown mechanism decreasing the permeability of the blood vessel wall.

It might be argued that the removal of the ciliary ganglion was not complete, or that accessory ganglia described by Anderson were still functioning. The paralyzing action of atropine would affect all the nerve endings, whereas mechanical removal of the ganglion might leave some endings untouched.

Our criterion of complete ganglion removal was the immediate and lasting effect on the pupil. In every case the pupil dilated after removal of the ganglion and remained perma-

nently widely dilated, and failed to constrict to either light or to eserine. We feel that this is sufficient proof that all the effective nerve endings had been destroyed.

Removal of the ciliary ganglion causes a decrease in the protein content of aqueous. It is extremely difficult to even hypothesize how this comes about. No explanation that we have to offer satisfies all of the experimental data which we have at hand. It is possible that further experiments will show that all of the changes in the protein content of aqueous which we have produced experimentally are due to the methods which are employed; for instance, the act of withdrawing the aqueous from the eye may in itself produce the changes in protein content when the iris is constricted or dilated by the drug being tested. Certain it is that we have no proof that the aqueous in such an eye has an alteration in its protein content prior to the time at which it was withdrawn from the eye. We should not, therefore, attempt an explanation of the various effects we have produced, until additional experimental evidence has been obtained. We can be certain of but one fact, namely, that removal of the ciliary ganglion does not prevent the increase of protein which eserine brings about. Hence eserine causes an increase in protein content by action on some other structure than the 3rd nerve fibres running to the ciliary epithelium. We are therefore unable to support Seidel's contention that the action of eserine is a proof of the secretory activity of the ciliary epithelium.

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A CASE OF AMYLOID DEGENERATION OF THE CONJUNCTIVA.

By Major R. E. WRIGHT, I. M. S.

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(*With two illustrations on Text-Plate XI.*)

E., HINDU, male, aged 36, was admitted to hospital on May 13, 1924, complaining of swelling of the lids and a discharge from the eyes which started three years before. On examination both upper and lower lids on either side were bulging and nodular, and very firm in consistence. The right cornea showed a small marginal area of exposure keratitis in the inferior temporal quadrant, the left cornea was clear. There was a slight mucopurulent discharge. The bulbar conjunctiva as far as could be seen was normal. On attempting to evert the lids it was found that with the utmost difficulty the upper lids could be partially everted, whilst the lower lids could not be everted at all owing to their rigidity. The mucous surfaces of the lids showed large irregular masses, smooth on the surface, with deep gutters between, the individual nodules being much larger and more irregular than seen in the grossest types of spring catarrh but without the irregular inflammatory thickening of the more massive types of conjunctival affections such as tuberculosis and Parinaud's disease. The fornices above and below respectively formed deep clefts bounded by rigid walls. The infiltration which caused this irregular thickening of the lids did not end at the fornices but encroached a little way under the peripheral bulbar conjunctiva. When seen in this position through the clear conjunctival membrane, the material infiltrating the subconjunctival tissues gave the appearance of being pink and fleshy, like lymphoid tissue, but solid in consistence. The masses in the palpebral conjunctiva were a dull brown red, and when the lid was squeezed between the fingers their consistence appeared to be that of a mod-

ILLUSTRATING ARTICLE OF MAJOR R. E. WRIGHT, I. M. S., ON "A CASE OF AMYLOID DEGENERATION OF THE CONJUNCTIVA."

erately soft wax. The layer overlying the degenerate tissue was thin and delicate and gave way with firm pressure of this sort. It was decided to remove the degenerate tissues of the lids underlying the epithelium, and to this end incisions were made in the depth of the fornices and the lids everted as carefully as possible. The tissue was then squeezed out with the fingers from between the tarsus and the superficial layers, its dislodgment being aided with a blunt scoop. With great care the lids were freed to a considerable extent and the remains of the conjunctiva, somewhat ragged but forming a good lining for the lids, settled back into position. The material thus removed reminded one rather of marrons glacés in appearance and consistence. It was plastic and practically avascular and could be molded between the fingers. Under the microscope it appeared to be made up of amorphous material irregularly broken up. This did not take the regular stains for amyloid except that in a very patchy way some scalelike and angular pieces showed up pink with methyl violet. There was very little cellular structure, blood vessels at infrequent intervals surrounded by a few round cells being all that remained. The stereophotograph shows the condition of the lids at the time of admission.

THE KNAPP ADRENALIN MYDRIASIS REACTION IN DIRECT DESCENDANTS OF PATIENTS WITH PRIMARY GLAUCOMA.[1]

By Dr. MARK J. SCHOENBERG, New York.

IN 1921, Dr. Arnold Knapp reported before the American Ophthalmological Society his observations on "The Action of Adrenalin on the Glaucomatous Eye," (1) and brought forth two important contributions to the problem of glaucoma: First, a diagnostic test for primary glaucoma; second, a convincing evidence that there is a connecting link between the pathology of the eye and that of the sympathetic nervous system in patients with primary glaucoma. The Knapp test has broadened the field of investigation for the glaucoma problem, and has suggested a number of questions worth while solving. During the past four to five decades there appeared in the ophthalmic literature a number of reports on cases of familial glaucoma, and the current opinion among ophthalmologists who have given the subject of glaucoma considerable thought is that heredity plays an important rôle in a certain group of cases.

Priestley Smith (2) in his well known series of articles on "Glaucoma Problems," says that a special liability to glaucoma may be inherited. Lawford (3) has published a number of examples of hereditary glaucoma illustrating the law of anticipation; *i.e.*, the tendency for the disease to appear at an earlier age in succeeding generations. Calhoun, (4) published a report on cases of hereditary glaucoma simplex. Nettleship (5) reported on cases of inherited primary glaucoma. R. H.

[1] Read before American Ophthalmological Society, Hot Springs, June 18, 1924.

Elliot, in his "Treatise on Glaucoma" (1922), speaks of "various modes of action of hereditary influences and of predisposition to nervous and vascular storms." Morax, in his book "Glaucome et Glaucomateux" (1921), says that heredity is a very important factor in glaucoma. Arlt, Mooren, Pfluger, Pagenstecher, Graefe, Kummer, Mules, Story, Jacobson, Lucien, Howe have reported a number of cases of familial glaucoma.

Well, if hereditary predisposition is an important factor for a certain number of primary glaucoma patients, it would be of no small value to determine, if possible, the incidence of this hereditary predisposition in the direct descendants of such patients. The question of potential glaucoma may be considered analogous, in a way, with that of the inherited predisposition to an infectious disease (tuberculosis, diphtheria, scarlet fever, measles, etc.). The potential glaucomatous, like the potential tuberculous or diphtheric, does not necessarily develop one of these pathologic conditions, but he or she may do so if the propitious circumstances are brought about. Of course, the discovery of this potentiality or predisposition has the great practical value of being the first step toward prevention.

There is no doubt that the Knapp adrenalin mydriasis test is positive in a very large majority of glaucoma patients. In his first series of sixty-five cases of glaucoma, sixty cases showed mydriasis after instillations of adrenalin in the conjunctival sac. My personal experience with this test confirms entirely Dr. Knapp's observations. Furthermore, he has found fifteen cases with glaucoma, apparent only in one eye, that the adrenalin instillation produced mydriasis in both eyes. (This is also confirmed by my own experience with the test in similar cases.) His significant remark that "susceptibility to adrenalin may be present long before the usual clinical signs of glaucoma." suggested to me the advisability of applying the adrenalin mydriasis test to the offspring of glaucoma patients—with the *purpose in view of finding out whether there is such a susceptibility to adrenalin among their direct descendants.*

The Knapp test is performed as follows: "After taking the tension with Schiötz tonometer and measuring the size of the pupil, one drop of adrenalin solution $\frac{1}{1000}$ (fresh P. and D.)

is instilled every few minutes for five times. After a half hour the measurements of the pupil and of the ocular tension are again taken." Many patients complain of a slight smarting and squeeze their lids after each instillation, thereby expressing much adrenalin. In order to obtain a more smooth application of the Knapp test, I found it practical to precede it by an instillation of one or two drops of one per cent solution of butyn or holocain. These anesthetics have no influence on the size of the pupil or tension of the eyes and present the great advantage of enabling us to instil the adrenalin drops and manipulate the lids in such a manner as to give a sort of a bath to the eyeball. In some patients with latent or confirmed glaucoma only one drop of adrenalin solution is sufficient to dilate the pupil in a short time to a marked degree; in others it takes five drops one entire hour to produce mydriasis. For practical purposes the positive adrenalin reaction is being called in this paper, one, two, three, four plus, according to the enlargement of the diameter of the pupil: + is when the diameter of the pupil enlarges by 0.5*mm:* ++ by 1*mm*, +++ by 1.5*mm*, and ++++ by 2*mm*, or more: ± when there was a doubt whether the pupil enlarged; − or negative when the pupils did not dilate at all.

So far thirty-five individuals, sons and daughters of thirteen patients with primary glaucoma, were submitted to the Knapp test. None of them showed any evidence of glaucoma.

The following is a brief report of the results obtained:

FAMILY I.—Mrs. F. N., age sixty-nine years. Right primary chronic glaucoma; cataract. Left, primary chronic glaucoma; optic atrophy, cataract. Son, S. N., age forty-two years. R.E. + 2.75 sph. = $\frac{10}{18}$, Jager 1; L.E. + 4.25 Sph. = $\frac{10}{16}$+, J. 1. Knapp test O.S.+. Daughters, (1) R.L., age forty-four years. R.E. + 3.25 sph., = $\frac{10}{16}$+; L.E. + 3 sph. $\frac{10}{16}$; add + 2.75 sph., Birkhauser 1.00. Knapp test O.U. ++. (2) H.R., age forty years. R.E. + 2 sph. = $\frac{10}{16}$+, add + 1.50, Birkhauser 0.6; L.E. + 75 sph. + 3.25 cyl, ax. 45, = $\frac{10}{16}$+, add + 1.50 sph. B.O. 6. Knapp test O.D. 4 drops ++++, irregular oval. (3) C. N., age thirty-five years. R.E. + 1.50 sph. + 25 cyl, ax. 180 = $\frac{10}{18}$, add + 75 Jager 1; L.E. + 1.50 sph. + .25 cyl, ax. 180 = $\frac{10}{18}$, add + 75 sph. Jager 1. Knapp test O.D. ++, twice tested 4 months apart.

FAMILY II.—Mrs. K. K., age sixty-four years. Primary chronic glaucoma O.U. states "several members of her family have glaucoma." R.E., + 2.50 sph. = $\frac{20}{30}$−, add + 3.00 sph. Jager 1; L.E. + 2 sph. = $\frac{20}{30}$−, add + 3.00 sph. Jager 1. Son, J. K., age twenty-nine years. R.E. = $\frac{20}{20}$; L.E. = $\frac{20}{15}$, Birkhauser, 1.5. Knapp test, ±.

FAMILY III.—Mrs. F. L., age sixty-two years. Primary chronic glaucoma O.U. Grandson, I. G., age eight and a half years. R.E. − 50 sph. − 50 cyl. ax. 90 = $\frac{20}{15}$; L.E. − 50 sph. − cyl., ax. 90 = $\frac{20}{15}$. Knapp test; neg.

FAMILY IV.—Mrs. R. P., age sixty-eight years. O.D. primary chronic glaucoma, O.S. absolute glaucoma. Sister has also primary glaucoma chronic O.U. Sons, (1) J. P., age forty-nine years. R.E. + 5.00 sph. = $\frac{20}{15}$+. L.E. + 4.50 sph. = $\frac{20}{50}$. Knapp test, after 2 drops both pupils oval, + + +. (2) L. P., age forty-eight years. R.E. + 2.50 sph. = $\frac{20}{30}$+. L.E. + 2.50 sph. = $\frac{20}{30}$. Knapp test, negative. Daughter, Mrs. Z., age thirty-eight years. Vision each eye = $\frac{20}{20}$. Knapp test, negative.

FAMILY V.—Mrs. R. L., age seventy-five years. Primary chronic glaucoma O.U. Son, F. L., forty-two years. R.E. $\frac{20}{15}$+, Jager 1. L.E. = $\frac{20}{15}$+, Jager 1. Knapp test, negative O.U. Daughter, L. K., age fifty-four years. R.E. + 75 cyl. ax. 180 = $\frac{20}{15}$; L.E. + 50 cyl. ax. 25, $\frac{20}{15}$, add + 2.50 sph., Birkhauser 0.7. Knapp test, + + + + O.U. regularly dilated (2 tests.) Granddaughter (mother's side), Miss L. K., age twenty-four years. Low degree of Hy. As. = $\frac{20}{20}$, O.U. Knapp test, negative.

FAMILY VI.—H. B., age forty-eight years. Primary glaucoma, chronic, O.U. Son B. B., aged twenty-three. Vision $\frac{20}{15}$ each eye. Knapp test +. Daughters (1), Miss M. B., age nineteen years. R.E. and L.E. = $\frac{20}{15}$, Knapp test + O.U. (2) A. B., age twenty years. R.E. and L.E. + 50 cyl. ax. 180 − $\frac{20}{15}$. Knapp test, negative O.U.

FAMILY VII.—Mrs. R. F., age forty-seven years. R.E., primary chronic glaucoma, L.E. acute primary, glaucoma. Sons, (1) H. F., age twenty years. R.E. + 1.75 sph. − 4.00 cyl., ax. 3.0 = $\frac{20}{40}$+. L.E. + 75 sph. − 2.75 cyl. ax. 176 = $\frac{20}{15}$. Knapp test, negative O.U. (2) N. F., age twenty-five years. R.E. + 1.00 sph. + 2.50 cyl. ax. 105, $\frac{20}{15}$; L.E. + 1.00 sph. + 3.50 cyl. ax. 75 = $\frac{20}{50}$. Knapp test, negative. (3) C. F., age twenty-four years. R.E. = $\frac{20}{15}$; L.E. = $\frac{20}{15}$ L.+ high myopic astigmatism. Knapp test, R.

+ + +; L. +. (4) M. F., age twenty-eight years. R.E. and L.E. = $\frac{20}{30}$+. Knapp test ±. (5) M. F., age seventeen years. R.E. and L.E. $\frac{20}{20}$−. Knapp test, negative. (6) S. F., age thirty-two years. R.E. and L.E. $\frac{20}{20}$. Knapp test, ±. Daughter, R. F., age eighteen years. Knapp test negative.

FAMILY VIII.—Mr. W. A., age forty-five years. R.E. primary acute glaucoma. L.E. primary chronic glaucoma. Son, L. A., age seventeen years. V. R.E. and L.E. $\frac{20}{15}$. Knapp test of R.E. and L.E., + + + +. Daughter, C. A., age eighteen years. V. R.E. and L.E. = $\frac{20}{15}$. Knapp test R.E. and L.E., ±.

FAMILY IX.—Mrs. A. G., age fifty years. Primary chronic glaucoma O.U. Sons (1) L. G., age twenty-nine years. V. R.E. and L.E. = $\frac{20}{20}$, slight My As. Knapp test, R.E. 4 drops, + + +; after 5 drops, + + + + regular; L.E. 3 drops, +, after 4 drops ++ pear shaped, eccentric. (2) Dr. H. G., age twenty-five years. R.E. + 50 sph. + 1.50 cyl. ax. 90 = $\frac{20}{30}$+; L.E. + 25 cyl. ax. 90 = $\frac{20}{15}$, Birkhauser, 1.5. Knapp test, negative. Daughter, P. G., age twenty-four years. R.E. = $\frac{20}{30}$, corneal opacity; L.E. = $\frac{20}{15}$. Knapp test, negative.

FAMILY X.—Mrs. I. M., age sixty-eight years. Primary chronic glaucoma O.U. Daughters (1) E. M., age twenty-eight years. R.E. + 50 sph. + 25 cyl. ax. 180 = $\frac{20}{30}$, Jager 1; L.E. + 50 cyl. ax. 180 = $\frac{20}{20}$+, J. 1. Knapp test, of R.E. + + +; L.E. +. (2) L. W., age thirty-four years. V. R.E. and L.E. = $\frac{15}{15}$. Knapp test, ± O.U.

FAMILY XI.—Mrs. G. H., age sixty-seven years. Right primary chronic glaucoma; Left, latent glaucoma (?) Daughters, (1) C. J., age thirty-nine years. R.E. + 50 cyl. ax. 180 = $\frac{20}{20}$. L.E. + 50 cyl. ax. 90, $\frac{20}{20}$. Knapp test, negative O.U. (2) Mrs. F. S., age forty-one years. R.E. − 1.50 sph. = $\frac{20}{70}$ (corneal opacities); L.E. = $\frac{20}{15}$. Knapp test, negative.

FAMILY XII.—Mrs. C. W., age forty-nine years. Primary chronic glaucoma O.U. Sons, (1) C. W., age thirty-two years. R.E. and L.E. = $\frac{20}{20}$. Knapp test, R.E., + + + ovale; L.E., + ovale. (2) A. W., age thirty-four years. R.E. and L.E. = $\frac{20}{20}$. Knapp test, R.E. +, L.E. ++. Daughter, (1) E. W., age twenty-five years. R.E. and L.E. = $\frac{20}{20}$. Knapp test, negative. (2) Mrs. R. B., age thirty years. R.E. and L.E. = $\frac{20}{20}$+. Knapp test, negative.

FAMILY XIII.—Mr. S. S., age fifty-four years. Right, primary glaucoma acute, left, chronic glaucoma. Daughter—I. S., age twelve years. R.E. and L.E. = $\frac{3}{10}$. Knapp test, R.E., + + +; L.E. +. Sons, (1) S. S., age nineteen years. R.E. and L.E. = $\frac{3}{10}$. Knapp test, + + O.U. (2) M. S., age twenty-five years. R.E., and L.E. = $\frac{1}{10}$. Knapp test, R.E. + + +, L.E., + +.

Of these 13 families, 15 daughters, 20 sons, 1 grandson, and 1 granddaughter, were examined by the Knapp adrenalin test. Of the 13 glaucoma patients 2 were male, and 11 female. Of the 15 daughters, 8 gave a negative Knapp reaction; 0±, 1+, 2++, 2+++, 2++++. Of the 20 sons, 6 were negative, 4±, 3+, 2++, 3+++, 2++++, one granddaughter, negative, one grandson, negative. The youngest daughter positive, was twelve years of age; the youngest son positive, was seventeen years of age. Of 15 daughters, 7 were distinctly positive and 8 negative. Of 20 sons, 10 were distinctly positive, 6 negative, and 4 doubtful.

In other words, about half were positive, and about 20 per cent were strongly positive, both among male and female descendants. There seems to be no marked predominance in either sex.

SUMMARY.

About 50 per cent of the direct descendants of the patients with glaucoma give a positive Knapp adrenalin reaction. This percentage will probably have to be revised as the number of cases tested will grow larger. The fact that such a large percentage among the descendants of glaucoma patients gives a positive Knapp reaction does not necessarily prove that they are going to be affected by the same disease. The paper merely establishes a fact, the interpretation of which will have to be postponed until more experience and study concerning this problem will be available.

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THE ORIGIN OF GLIOSIS RETINÆ IN CASES OF STAPHYLOMA CORNEÆ.

By Dr. Peter S. Soudakoff, Peking.[1]

(With four illustrations, two in color, on Text-Plates XII. and XIII.)

In a case of staphyloma of the cornea, we find, as a rule, that the retina is not very much affected in the beginning. There may be present merely slight symptoms of inflammation (perivasculitis) due to an irritation produced by the ulcer of the cornea. Later, secondary glaucoma sets in and destroys the vision. The ganglion cells and the nerve fiber layer disappear, due to an atrophy of the optic nerve brought about by the glaucomatous excavation.

In the histological study of advanced cases of staphyloma of the cornea we often find, besides such an atrophy of the retina due to the glaucoma, other changes. The retina becomes thin, the rods and cones disappear, and in places the two nuclear layers are fused into one. The tissue is more intensely stained and more compact. This condition of the retina is described by Leber (1) as "gliosis of the retina."

In certain cases a proliferation of the pigment epithelium is also present and the pigment cells are scattered irregularly in the degenerated retinal tissue. Leber differentiates this from gliosis as the "secondary pigment-degeneration of the retina."

These pathological changes are also of clinical interest, because in exceptional cases such a condition in the fundus can be seen, when the staphyloma is only partial and an ophthalmoscopic examination is possible.

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ILLUSTRATING DR. PETER S. SOUDAKOFF'S ARTICLE ON "THE ORIGIN OF GLIOSIS RETINÆ IN CASES OF STAPHYLOMA CORNÆ."

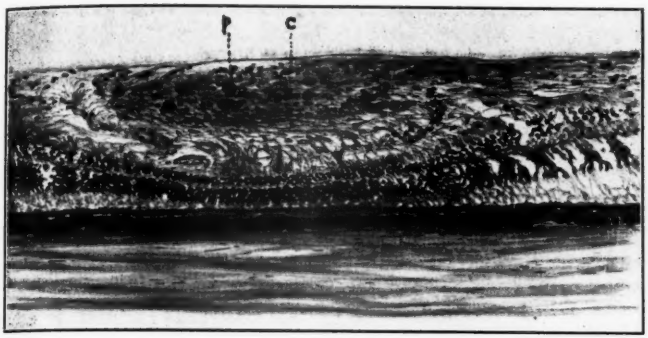

FIG. 1 × 150. The spindle shaped cells with oval nuclei are accumulated in the innermost part of the nerve-fiber layer (c). Blood pigment lies between the cells. The inner nuclear layer is thin. The rods and cones are reduced to half the normal size. The two nuclear layers are bent around the focus and lie against the pigment epithelium.

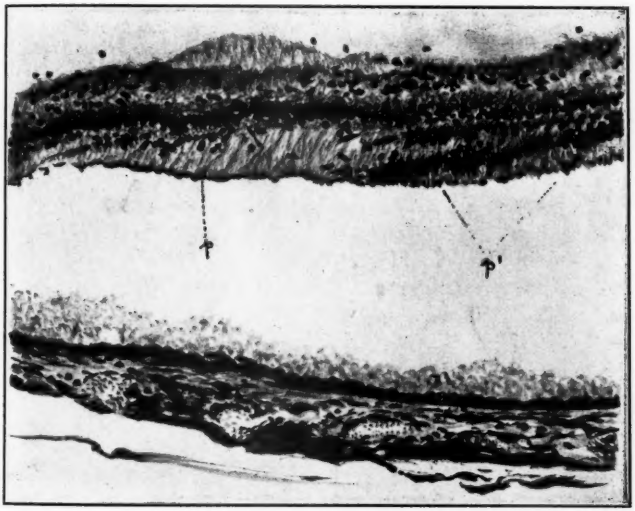

FIG. 4. A focus of atrophy in the outer layer × 150.

The nerve-fiber layer is atrophic. The inner nuclear layer is represented by only a few rows of nuclei while the outer nuclear layer is almost completely destroyed—only a few cells remain. The rods have disappeared. A line of pigment, attached to the outer border of the retina (p) and the pigment on the adjacent retina (p^1) are due to an artificial detachment.

ILLUSTRATING DR. PETER S. SOUDAKOFF'S ARTICLE ON "THE ORIGIN OF GLIOSIS RETINÆ IN CASES OF STAPHYLOMA CORNEÆ."

FIG. 2. Blood pigment in the retina stained with Prussian blue and Alum Carmine, × 70.

The nerve-fiber layer is rich in oval nuclei and contains clumps of blood pigment which are stained blue. The two nuclear layers show no pathological changes except slight blue staining in places. The rods and cones are apparently intact.

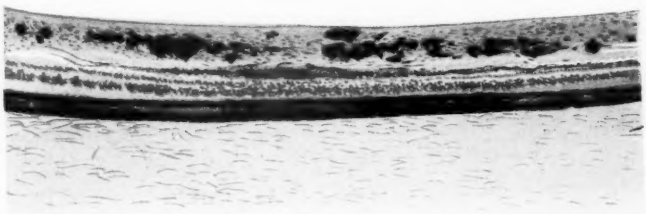

FIG 3. Focus with blood pigment and a fusion of the nuclear layers. Section stained with Prussian blue and Alum Carmine, × 70.

The nerve-fiber layer is sclerosed and is full of blood pigment. The tissue between the nerve-fiber layer and the inner nuclear layer is bluish, being impregnated with iron salts. The two nuclear layers are fused into one. The rods and cones are totally destroyed.

E. Fuchs (2) in his two papers on retinal changes in cases of staphyloma, concludes that there is no difference between gliosis and secondary pigment-degeneration. The two conditions are more or less identical except that in gliosis the pigment epithelium does not proliferate and in secondary pigment-degeneration the pigment epithelium proliferates and migrates into the retina.

According to him a gliosis, with or without pigment, is produced first, by a slight inflammation so commonly seen in the retina of a staphylomatous eye, second, by a secondary glaucoma, and third, by a certain displacement of the retina.

This displacement is produced as follows: the ora serrata is often connected with the lens by means of very fine connective tissue fibers, or with the staphyloma if the lens has been expelled at the time of perforation of the cornea. These fibers are due to the presence of a slight exudation in the vitreous caused by an inflammation in the anterior segment of the eye. The latter becomes ectatic; traction is exerted through these fibers on the ora serrata. As a result of this the retina is drawn forward over the pars plana of the ciliary body.

This is called by him a "supertraction of the retina." The retina does not become detached because it is held in position by the increased pressure in the vitreous body (secondary glaucoma). In such cases the retina merely glides over the choroid, thereby producing a displacement of the rods and cones which consequently are destroyed.

It seems therefore that changes in the outer layer of the retina due to a supertraction produce chiefly a gliosis, while atrophy of the inner layers is caused by a glaucomatous cupping of the nerve.

The following case is reported because, first, the changes in the retina reveal another etiologic factor of gliosis, and second, such pathological changes, if seen clinically, may simulate choroiditis.

CASE REPORT. No. 6058.—A girl from one of the famine districts, age 6, has been blind in both eyes for two years due to an attack of purulent ophthalmia. The eyes were not treated. The right eye gradually became atrophic while the left became enormously ectatic. It was on account of the disfigurement that the patient was brought in

for treatment. The patient was admitted on September 17, 1923, and the left eye was enucleated on the following day.

PATHOLOGICAL EXAMINATION.

Eye ball was fixed in 10% formalin for 5 days. It was sectioned through its horizontal meridian.

Macroscopic Examination.—The eyeball is enlarged. It measures antero-posteriorly 33*mm*, and horizontally 23*mm*. The cornea is almost entirely replaced by a scar and the vitreous is connected with the posterior surface of the staphyloma by a membrane. The entire retina is covered by numerous spots more or less as large as the disk. These spots closely resemble foci of choroiditis and are of different colors: yellow, brown as if they were pigmented, and whitish or pale with a brownish center. The macular region appeared intensively yellowish at the time the bulbus was sectioned but disappeared in 24 hours after immersion into 80% alcohol.

Microscopic Examination.—On the nasal side of the staphyloma some corneal tissue is recognized and is very much attenuated. Behind it the iris is compressed into a thin intensely pigmented layer. Elsewhere the iris has disappeared and only traces of its pigment epithelium are found scattered behind the very vascular staphylomatous scar. The position of the former pupil is recognized only by the fact that behind the staphyloma there is an eccentric area in which the pigment epithelium is wanting.

The vitreous body is shrunken and is adherent to this pupillary area. It extends from the ora serrata to the posterior surface of the staphyloma and appears as a reddish membranous shred.

The anterior chamber is absent but the posterior chamber is enormously distended. The lens is absent.

The ciliary muscle is not recognized; the ciliary processes are atrophic and are displaced toward the staphyloma.

The sclera is everywhere attenuated, its intercalary region is somewhat ectatic (intercalary staphyloma).

The disk has a deep excavation with an overhanging margin. Bending over the margin of the excavation some nerve fibers are recognized. They are densely intermingled with the nuclei of the neuroglial fibers.

The fibers of the optic nerve have lost their regular arrangement and are atrophic. Only a few bundles of edematous nerve fibers are seen on the nasal side of the periphery of the nerve.

The retina is not detached except in a small area near the

posterior pole. It is a slit-like detachment. Two distinct pathological changes are found in the retina as follows:

I. *General.*—A general change, namely, the ganglion cells and the nerve-fiber layer have mostly disappeared while the inner nuclear layer has become vacuolated. Müller's sustentacular fibers are clearly visible. The outer nuclear layer is poor in cells which have lost their regular arrangement, having wandered into the outer plexiform layer. The rods and cones are present except at certain places which will be mentioned later. The pigment epithelium appears normal and contains the usual amount of pigment.

Besides the above mentioned changes we find other special changes:

II. *Special.*—1. In the inner layers of the retina there are small foci of spindle-shaped accumulations of cells with oval nuclei; the length of each focus is about 0.8*mm*. (Fig. 1.) These cells are scattered over densely stained reddish tissue. They are probably neuroglial cells. The accumulations of cells are separated from the inner nuclear layer by very loose tissue. In certain sections remnants of the nerve-fiber layer are visible in this loose tissue. This proves conclusively that the accumulations of cells are within the nerve-fiber layer. The foci contain not only cells but also some yellowish-brown pigment. Some of the pigment is in the form of large clumps, probably phagocytosed pigment, and some appears as very fine granules. Where a focus is found the two nuclear layers are bent around it as if they have been pushed against the pigment epithelium. Here the inner nuclear layer is thinner than that away from the focus while the outer nuclear layer is of normal thickness. The rods and cones are reduced to half their normal size. The curve described by the two nuclear layers is, as a rule, about twice the length of the focus.

Sections stained with Prussian blue show further interesting details. Each focus contains some dark bluish granules surrounded by a light blue halo. They are of various sizes. The close contact of the halos renders the entire focus bluish. The dark bluish granules correspond to the pigment mentioned above. In addition, very fine light bluish spots are also seen between rods and cones. Hence the foci contain various amounts of iron salts as well as pigment and these foci correspond to the small brown spots seen macroscopically.

2. Near the posterior pole of the retina the largest foci are found. They are about 1.2*mm* in length. Their structure is more or less similar to that of preceding foci but two other pathological changes are recognized.

(a) At places where the foci are found the nerve-fiber layer has become denser and thicker, consequently, the retina appears thickened. (Fig. 2.) In the nerve-fiber layer there are many nuclei. The two nuclear layers and the rods and cones show no pathological changes. The foci are diffusely and deeply stained with Prussian blue in which there are found a few pigmented corpuscles. The pigment epithelium here, as elsewhere, is normal.

(b) In contrast to the changes in (a) some of the foci are accompanied by a marked attenuation and bending of the retina (Fig. 3). The curve described by the retina encloses the focus and measures about 2.0*mm*. In these foci the fibrous elements are quite dense, sclerosed, and contain numerous nuclei. Pigment is found only in the center of these foci. In sections stained with Prussian blue, not only the pigmented corpuscles are stained bluish, but also the very narrow strip of tissue lying between the proliferated nerve-fiber layer and the inner nuclear layer. Unlike the preceding, in which practically only the nerve-fiber layer is affected, the two nuclear layers are fused into one, and the rods and cones are almost entirely destroyed. It is to be noted here that the area of pigment within the foci is quite small as compared with that of the fibrous tissue. In the periphery of the foci the anatomical picture of gliosis of the retina is also clearly demonstrable. The blood vessels in the vicinity of the foci seem to be changed also, for we find in several sections thrombosis of the retinal vein. The entire pigment epithelium of the retina appears normal.

Undoubtedly the two types of foci correspond to the pale spots with a brownish center described macroscopically.

3. These foci are not quite so large as those described above (2). Within the foci the retina is markedly atrophied. The nerve-fiber layer shows no increase of nuclei or fibers. The two nuclear layers are fused into one and their nuclei are considerably diminished in number. Their outline is not sharply defined because, away from the foci, the nuclei gradually increase in number and are separated again into two distinct layers. The external limiting membrane is clearly visible but the rods and cones have entirely disappeared. The pigment epithelium appears intact.

Within the walls of a few small blood vessels there are fine particles of pigment which appear darker than those within the foci and do not show an iron reaction. The pigment particles in the nerve-fiber layer likewise do not possess such a reaction. It seems therefore that these particles of pigment within the retinal vessels are derived from the pigment epithelium. It is remarkable to note that there is no synechia anywhere between the rods and cones—and in

their absence the external limiting membrane—and the pigment epithelium.

These foci represent typical foci of gliosis (3) and correspond perhaps to some of the pale spots seen macroscopically. Since they resemble in structure the foci described in 2 (b) there is no doubt that they are also caused by hemorrhages. They are perhaps older and the blood pigment has been totally absorbed.

4. In only one of the sections examined is a focus of accumulated pigment cells found in the outer layers of the retina. This focus lies approximately 4*mm* behind the ora serrata, and occupies a space of 0.1*mm*. It is composed of cells filled with pigment which are stained with Prussian blue. Only a few of these cells are not stained by this method. They are probably cellsd erived from the pigment epithelium in spite of the fact that the latter appears quite normal and does not show any proliferation. This accumulation of cells appears to be lying upon the pigment epithelium and produces a defect in the two nuclear layers.

As it is impossible to find any foci in all the sections examined corresponding to the yellowish spots mentioned above, we are led to believe that these spots represent an intermediary stage of development between the yellowish brown and the whitish or pale spots.

Discussion.

This case of staphyloma is of special interest for two reasons.

First.—The spots which appear macroscopically like foci of choroiditis are due to changes principally in the nerve-fiber layer of the retina. Such changes are undoubtedly caused by hemorrhages. This is proved by the fact that most of foci are impregnated with iron salts and are diffusely stained by Prussian blue. We know that under certain conditions hemorrhages in the retina may produce a clinical picture of retinochoroiditis when there is a destruction of the retina, as seen in the cases of retinitis sclopetaria (4) as a result of a contusion produced by a gun-shot passing through the orbit. The fundus in such a case is covered with numerous black, and white spots. The former are due to a proliferation of the pigment epithelium and the latter are due to an atrophy of this epithelium with the formation of white membranes in the retina. As a rule, after the condition has existed for some time, no blood pigment can be found in the retina microscopically.

Retinitis sclopetaria presents the ophthalmoscopic picture of a wide spread choroiditis. But such a fundus picture would probably be different from that of the present case of staphyloma, had an examination of the latter's fundus been possible.

Certain clinical cases of retinochoroiditis are produced by deep hemorrhages in the retina, in which small defects of pigment epithelium appear as sharply outlined reddish or yellowish spots surrounded by a small amount of pigment. The retina of the present case of staphyloma shows such spots macroscopically but they are not surrounded by pigment.

The reason why blood pigment has accumulated in the center of the foci in this case while it does not in an ordinary case of retinal hemorrhages may be explained as follows.

Common retinal hemorrhages disappear, as a rule, by gradually becoming smaller and at the same time fainter and paler. Exceptionally, we see white spots in the center of a hemorrhage in a case where fatty degeneration of the nerve-fiber layer is present.

An inflammation of the retina may be the cause of a retarded resorption of the retinal hemorrhage which in turn may produce a different reaction by the tissue, for in the pathological sections examined, we found a sclerosis of the nerve-fiber layer, compression and atrophy of the two nuclear layers, and an accumulation of phagocytosed iron-pigment in the center of each focus.

Likewise we find, pathologically, a retarded resorption of blood in the anterior segment of an eye which has been inflamed. Furthermore when blood remains for a long time in the anterior chamber of an eye with iritis, we not infrequently see covering the iris and filtrating angle, a great number of phagocytes filled with blood-pigment (3).

Hemorrhage in the supra-choroidal space of an inflammed eye very often becomes encapsulated by connective tissue in which a variable amount of blood-pigment is found.

We believe that hemorrhages in the retina of an inflamed eye due to the perforation of a corneal ulcer with iris prolapse, are similar to those just described. Moreover, they may cause pigmentation of the retina with proliferation of the neuroglia as in a case of hemorrhagic cysts of the brain.

Second.—Another interesting feature in this case of staphy-

loma is that at certain places where the nuclear layers have been compressed by the hemorrhages, the retina presents a picture of gliosis. If there were no blood pigment found in the foci, we would have a typical picture of gliosis, namely, destruction of the rods and cones, fusion of the two nuclear layers, and attenuation of the retina.

We see, therefore, that a pathological picture, similar to a gliosis, may be produced by hemorrhages in the nerve-fiber layer of the retina. Hence a gliosis may develop in the nerve-fiber layer which is an "inner layer" of the retina. But in the opinion of E. Fuchs and Leber, gliosis is usually caused by changes in the "outer layer" of the retina.

Undoubtedly we often find in the outer layer of the retina, pathological changes which may be considered as the origin of gliosis. The following case is an illustration:

CASE 3390.—Miss Y. S. T., age 15 was admitted to the St. Luke's Hospital, Shanghai, for enucleation of her right eye on account of total staphyloma of the cornea. The vision was nil. Enucleation was performed by Dr. Owen Han and the specimen was sent to the Eye Department of the Peking Union Medical College for examination.

Macroscopic Examination.—Cornea is staphylomatous and shows at its lower part a xerotic area. Above there is a small black spot. Vertical section on the temporal side is made and a segment is removed. The lens is obliquely subluxated and inclines backward. At the center of the posterior surface of the staphyloma, a small grayish lentil-shaped translucent mass is found.

Microscopic Examination. Cornea.—The central portion of the cornea is replaced by a staphylomatous scar which is rich in vessels and lymphocytes. Vessels from the limbus grow toward the center of the cornea. The epithelium is normal but in certain sections it is wanting, due probably to exposure of the eyeball. Some of the sections show a cystic scar with numerous small epithelial cysts. Bowman's membrane is largely destroyed. Descemet's membrane is found only in places where the anterior chamber remains unobliterated. The anterior chamber is divided into several narrow slit-like chambers by the synechiæ.

Iris.—The pupillary margin of the iris is almost totally prolapsed and is transformed into a scar tissue. In certain sections the sphincter of the iris is still recognizable. The ciliary portion of the iris is adherent to remnants of the cornea and is atrophied. The filtration angle is blocked by

the root of the iris and the pectinate ligament is nowhere to be found.

Ciliary body.—The ciliary body itself does not show any change but its processes and the unpigmented epithelium are quite thin and atrophied.

Lens.—The lens shows a beginning secondary cataract near anterior pole.

Sclera and Choroid.—Normal.

Retina.—The retina shows the following changes. The nerve-fiber layer is almost totally gone, only the Müller's sustentacular fibers remaining. A number of sclerosed vessels are found. The walls of the arteries are thickened while those of veins are infiltrated with lymphocytes. The lumen of some of these blood vessels is obliterated. Minute foci of hemorrhages are found in the different layers of the retina. In the ganglion cell layer the cells are very few in number. The inner nuclear layer is everywhere preserved but in certain places it is rather attenuated. The outer nuclear layer and the layer of rods and cones appear normal but in the anterior part of the retina it shows some special changes.

1. In the vicinity of the ora serrata some minute adhesions between the retina and the pigment epithelium are found which undoubtedly are caused by foci of inflammation.

2. About 4*mm* behind the ora serrata, there are places where the inner nuclear layer is represented by only a few or even a single row of nuclei with an almost complete destruction of the outer nuclear layer—only a few nuclei are seen in the meshes of Müller's fibers (Fig. 4). Here the rods are wanting and a line of pigment is attached to the outer border of the artificially detached retina. There is no sign of there having been a direct adhesion between the retina and the pigment epithelium where these changes are found, because the pigment on the retina, at the focus, is not any greater in amount than the pigment on the adjacent retina.

The cause of such changes is not clear but the fact that there is no adhesion between the retina and the pigment epithelium is a proof against an inflammatory origin. It is very likely that the hemorrhages in the outer layers of the retina had produced a destruction of these layers and a decrease in the number of their nuclei.

CONCLUSIONS.

The two cases of staphyloma of the cornea presented in this paper prove conclusively that hemorrhages play an important

rôle in the etiology of gliosis of the retina and should be considered as a significant factor.

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HETEROCHROMIA IRIDIS.

By Dr. F. Herbert Haessler, Milwaukee.

IT has long been recognized that in most people the irides are alike in color, but that occasionally they differ in this respect. It was tacitly assumed that a difference in color was merely a natural peculiarity without significance or in need of explanation, until Fuchs pointed out that a low grade, chronic cyclitis and cataract frequently occurred in the lighter colored eye.

With his usual clarity and precision, Fuchs noted all essential manifestations of the disease in his first communication. In these cases the heterochromia occurs in blonds as well as brunettes. The difference in color of the irides is usually observed early, though occasionally a patient insists that the color of one eye has gradually become bleached. Often a patient points out that one eye is like that of the father and the other like that of the mother. There is no pain; no sign of inflammation; no demonstrable association with general, bodily disease. The patient usually comes to the oculist because of defective vision, the cause of which is discovered to be cataract, which Fuchs found present in 33 of 38 cases.

As is frequently the case in cataracta complicata, the opacity begins in the posterior cortex. When complete the lens is bluish white with distinct sectoring and a great tendency to swell.

Despite the fact that the cataract is a complicated one, extraction is easy and the prognosis good. The nucleus is usually small and surrounded by a large amount of soft cortical tissue. The nucleus remains white and without sclerosis, even at an age when sclerosis of the lenticular nucleus is normal. In an an occasional case lenticular opacity is absent

and these cases may be more common than experience leads one to suppose, because the difference in iris color, alone, without a disturbance of vision, does not often cause the patient to seek advice.

Precipitates on the posterior surface of the cornea are almost constantly found. They are very fine, usually limited to the lower one half of the cornea and, because of their small number, are easily overlooked. They are not pigmented and never confluent. The aqueous is always clear and no large mass of exudate is ever found in the dependent part of the chamber angle. The iris differs characteristically from a normal light iris. While normally the tissue seems transparent, so that occasional vessels are clearly seen and the retinal pigment layer shimmers through the thinner areas, as for instance the crypts, in a heterochromic eye the tissue of the iris obscures these details so that they seem hazy or masked. Often, however, these differences must be sought with a loupe. Marked atrophy of the iris and fresh foci of inflammation are always absent. The retinal pigment layer of the iris is frequently defective, so that the normally black margin of the pupil is ragged, narrow and gray.

Occasionally the pupil of the affected eye is larger than the other. Fuchs attributes this to atrophy of sphincter fibers.

Fine vitreous dust is not rare. The very faintest ciliary injection has been observed on rare occasions.

The disease begins insidiously and lasts for decades. There have been many guesses as to the etiology, but it has been impossible to show that any of the common infections causing chronic inflammation are the cause of heterochromia. Fuchs, himself, after studying several enucleated eyes affected with the disease, has corrected his earlier description of the pathological picture which was based on a study of bits of iris obtained by iridectomy. In his sections it is clear that the color of the iris is due to a paucity of normal chromatophores. There is no evidence whatsoever of destruction of chromatophores which previously existed. Therefore, one point is definitely settled. The light color of the iris is not the result of inflammatory changes. An as yet unknown etiological agent must be active in earliest childhood or perhaps during intrauterine life. The inflammation is manifested by a very

slight diffuse infiltration of the uvea with lymphocytes, plasma cells and occasional eosinophiles and mast cells. Whether the nests of epithelioid cells found in one of his cases and in one reported by Schlippe, are part of the picture, must be determined by examining a large series of cases. The extent of the inflammation is much greater than the clinical examination leads one to suppose. On the surface the inflammation manifests itself by the precipitates on Descemet's membrane and proliferation of the ciliary epithelium, as in other chronic uveitis. Exudation is never found, nor does it in spite of its long course produce severe tissue changes in the uvea. The fact that the lens is often clear in much severer uveitis suggests that another factor must enter into the causation of the cataract—possibly a developmental anomaly of the lens itself. The frequent presence of congenital anomalies of uvea and retina makes if not unlikely that the lens is also congenitally less resistant to noxious agents.

It is to this category of heterochromia that my cases belong.

CASE I.—W. S. B. White, male, aged 24, painter. Cataract had been discovered when patient was examined as applicant for employment.

With naked eye iris of right eye is green, that of left eye distinctly bluish. The eyes are free from hyperæmia. The right eye is entirely free from abnormal manifestations. The ophthalmoscope reveals nothing but opacity of the left lens. With the slit lamp many fine, dew-like deposits are seen over the posterior surface of the cornea, as well as small and large precipitates, none of which is pigmented. The aqueous humor is clear. The iris tissue differs from the right, only in that it is slightly less translucent. The anterior lens surface is covered with fine, colorless debris. Outer cortex has fine, radial striations and some fluffy, white, fine opacities seen chiefly in the peripheral ends of the radii, 0.1*mm* in diameter. The lens nuclei are clear. The posterior cortex is like the anterior, except at the posterior pole, where a knob of honeycomb-like, opaque lens tissue projects into the otherwise clear cortex. The superficial layer of the posterior, cortical zone is completely opaque and displays many areas of iridescent color.

Three months later no change had taken place, except that the iridescent coloring was no longer demonstrable, suggesting that the posterior, cortical opacity had become thicker. Five months after last examination cataract is

found to have progressed. Club-like opacities lie in the anterior cortex, extending from periphery into pupillary area.

CASE 2.—E. W. White, female, aged 51. Right eye has been green, left blue, as long as she can remember. Remembers no disease of eyes at any time other than cataract in left eye, noted at about 32 years of age.

Examination grossly and with slit lamp. Right eye free from abnormalities. Iris green with scattered areas of brown pigmentation, occasional visible radial blood vessel and entirely intact retinal pigment layer, as determined by transillumination and inspection of its visible margin at pupil.

O. S. Cornea clear except for precipitates on its posterior surface. Here are found dew-like deposits, amorphous dust, white single cells in clumps and stars. No pigmented precipitates were found. The iris is blue. Occasional radial blood vessels are seen as in O.D. The tissue is slightly less transparent than normal, and dull. Areas of brown pigmentation are entirely absent. Except in a few areas the pigment layer is entirely invisible at the pupillary margin and in these areas it is rarified. Transillumination reveals large clefts in this membrane, extending in from the pupillary margin from 1 to 3*mm.*

The lens is entirely opaque to the very surface of the cortex and many large clefts are visible.

Both of these cases exhibit the cardinal symptoms—heterochromia, cataract and precipitates.

In Case 1 the cataract was posterior capsular and in Case 2 total. In both the precipitates were small, numerous and unpigmented. Dew-like deposits were seen in both cases. In Case 2 there was atrophy of the retinal pigment layer.

Dew-like deposits constitute a sign of eye disease revealed only by the slit-lamp and are an expression of œdema of the endothelium of Descemet's membrane. They are found in corneal and intraocular inflammation, glaucoma and other intraocular diseases. The dew-like deposits are a physical sign not observed in heterochromia before the use of the slit-lamp. They throw no new light on the etiology, nor do they give added weight to any one of the theories which have been advanced to explain pathogenesis. The obscurity of the etiology has led to much speculation. Of course, cases of

difference in iris color produced by hyperæmia, siderosis and atrophy from iritis and glaucoma, are not a part of the heterochromia problem.

In seeking to explain the phenomena of essential heterochromia, Fuchs postulates an as yet unknown etiological agent acting in earliest childhood or perhaps in intrauterine life, and bases this on the demonstrated fact that the paucity of pigment precedes the complications, and that a pigment atrophy consecutive to the complications need not be considered.

Scalinci attributes all changes of heterochromia to bilaterally asymmetric tone of the sympathetic nerves, due to a lesion in the cervical sympathetic ganglia, the heterochromia being a trophic disturbance in one iris. He bases his theory on the fact that in many cases of heterochromia a difference in size of pupil, of lid slit and relative enophthalmos is observable on the side of the lighter eye. Scalinci points out that we must not consider the presence of precipitates an absolute proof of the existence of cyclitis, and believes that the low sympathetic tone makes the blood vessels more permeable for cells.

It has been objected against Scalinci's theory that manifestations of asymmetrical sympathetic tone, though common in simple heterochromia, are very rarely observed in heterochromia complicated by precipitates and cataract. Furthermore experimental excision of the cervical sympathetic has produced a lighter iris in only one doubtful case, though the experiment has been performed many times by many observers. Clinically a depigmentation of one iris following injury to the cervical sympathetic, is not recorded. It is the consensus of opinion among competent observers, that to be effective the sympathetic lesion must exist in the embryo before the mesodermic layer of the iris has developed.

Streiff has elaborated a much more complex explanation first suggested by Lutz, but he bases some of his statements on approved assumptions and postulates mechanisms of pathogenesis not definitely known to exist. He distinguishes heterochromia simplex and complicata. In the former one eye as a whole is inherited from the father and the other from the mother; the hereditary determinants (Vererbungs einheiten) were completely crossed. In complicated heterochromia he assumes that the normal eye is inherited entirely

from one parent, while the lighter eye is inherited chiefly from one parent, but the iris color is determined by hereditary determinants (Vererbungs einheiten) from the other parent, and in the course of development these ambivalent hereditary determinants (Vererbungs einheiten) are incompatible. From the incompatibility a vasomotor disturbance arises, producing precipitates and cataract. The fact that other congenital anomalies are often associated with heterochromia gives weight to the idea that the heterochromia problem is one of heredity. To support his theory Streiff cites a piece of work by Steiger, who after many years of observation concluded that usually eyes are inherited as a whole, but that a "mosaic inheritance" does occasionally occur. However, precise information regarding asymmetrical inheritance of paired structures is very incomplete, and it seems unlikely that a mixture of inheritance determinants should produce pathological changes in the eye any more than in the body as a whole.

It is v. Herrenschwand's opinion that there are two kinds of heterochromia, namely the comparatively rare complicated heterochromia and the rather frequently seen heterochromia simplex. For the former he accepts the explanation of Fuchs and believes that the latter is a manifestation of bilaterally asymmetrical sympathetic tone. He has collected at least 13 cases in the two years in which he been looking for them. In each case the eyes were normal aside from a paler iris on the side on which there was also a smaller pupil, narrower lid slit, slight enophthalmos or greater filling of conjunctival vessels. In some there was decreased eye tonus on the affected side, or unilateral blushing and sweating.

I have recently observed two cases in which there was anisocoria and a slightly paler iris on the side of the small pupil. It is, however, not necessary to assume that it is the difference in sympathetic tone that produced the color difference. Münch believes he has shown that in the monkey embryo the anterior iris layer contains not only stroma cells, but a thick layer of primitive ganglion cells, such as he associates with sympathetic nerve endings. If it is also true in man that stroma cells and sympathetic cells develop together, then it is easy to understand that in a lighter eye there will be a smaller pupil, merely because there are less dilator fibers stimulated.

SUMMARY.

Heterochromia exists as an entity characterized by a pale iris in one eye, complicated in many cases by precipitates and cataract. The light color of the iris results from a failure of chromatophores to develop and not from atrophy of chromatophores already in existence. The complications are not the cause of the disappearance of the chromatophores, hence the usual etiological agents of iritis, such as syphilis and tuberculosis, are not the cause of the color difference of the irides.

The prognosis is good. Inflammatory manitestations never become severe and the cataract can be extracted without difficulty.

The etiological agent is unknown, but it must become active very early in life and probably in the embryo.

It is probable that a second group of cases exists, in which the difference of iris color is associated with unilateral sympathetic hypotone. These cases remain free from precipitates and cataract.

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CIRCUMSCRIBED LAMELLAR LENS CHANGE PROBABLY INHERITED.

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VOGT has mentioned, among his exhaustive studies of lens abnormalities, the following as unique. A lady, 37 years old, at the ages of 10 and 17 years suffered a parenchymatous keratitis. Today irregular corneal scars, which reach the Descemet membrane, are visible, also deep vessels. Her father, 70 years old, shows extensive equatorial chorioiditis of long standing in both eyes. The patient's lens presents a pronounced white to red yellow lamellar zone of opacification, in the posterior axial region, on the border between central and posterior thirds of the lens. The opacity lies in a discontinuity zone, which can be traced around the lens-equator region and anterior to it. The opacification therefore does not reach to the equator. In the right lens the condition is somewhat similar, with the exception that the opacity more nearly approaches the equator. The posterior lenticular surface is relatively less convex, while the anterior surface is more so than is usually found in normal eyes.

Vogt does not know how to explain this condition. He is inclined to think there is a connection between the parenchymatous keratitis and this layerlike opacity. Its deep location, however, speaks against this view. He expresses the hope that further observations of similar cases may aid in solving this question. The following observation may not solve the mystery, but it will show that a supposed dependence on a corneal process was only a coincident feature of Vogt's first observation.

The patient, 32 years old, born in Alsace, has some asthen-

opic complaints. V. each eye $\frac{6}{6}$ with cyl. 0.5 D, with both eyes $\frac{6}{4.5}$. The right cornea presents a central corneal macula. Fundi, and other media, are normal. With dilated pupil, using a 100 watt oblique illumination, a disk-like shadow is visible, deep within the pupil. This shadow, which has an apparent diameter of 4 to 5*mm*, cannot be seen with the +10 D lens and mirror in focal light. The Gullstrand nitra-lamp shows the relief image of the adult nucleus well defined, and more than usually pronounced for that age. Within the adult nucleus a distinct yellow, cloudy, ring is seen, which surrounds the embryonal nucleus. The top and bottom of this ring seem broadened, which latter I accept as an expression of the more or less oblique section. The anterior and posterior lamellæ of this ring are not seen equally distinct at the same time. They must be individually focussed. The yellow color surrounds the nucleus entirely; when moving the lamp the ring is seen to become smaller toward the periphery, until the entire space within the band becomes yellow at its poles, as one would expect if it surrounded a globular body.

Below the lower point some small punctate-opacities are found in the cortex. The erect and inverted Y sutures, are distinctly visible, with an indication of fibrillar radiation. The clear interval in the lens center is also distinctly visible.

The vitreous contains many threads and more small dots than are normally found. These latter disperse the light. Directly behind the lens the reflex of the vitreous tissue is compact and arranged in a curtain-like manner. The circumscribed location of the phenomenon suggests a physical cause for the yellow tint, comparable to the coloring of the whole of the lens, which latter increases with age, rather than that a true pigment may be present.

The condition is surprisingly alike in both eyes, even including the group of small dots in the lower cortex. With an undilated pupil the ring cannot be seen in its entirety.

The highest magnification fails to show formed elements within the ring. The patient was seen two years ago (1921) and again in the summer 1923 and the condition had not changed.

Recently I had an opportunity to examine the patient's

mother, 67 years old. V. each eye $\frac{6}{6}$. A form of coronary cataract was found with broad palisades which meet, and thus give an impression of cuneiform cataract. Besides many dots and spots in the cortex there is a suggestion of a yellow band, especially visible in the left lens. This yellow zone is only visible when one searches for it, and therefore a psychic factor of expectation of the examiner to find it cannot altogether be excluded. The lenses have fissures filled with fluid, especially in the right eye.

See A. Vogt, *v. Graefe's Arch. Bd.*, 109, p. 200.

REPORT OF THE PROCEEDINGS OF THE SECTION ON OPHTHALMOLOGY OF THE NEW YORK ACADEMY OF MEDICINE.

By Dr. DAVID H. WEBSTER, Secretary.

MEETING OF NOVEMBER 17, 1924. DR. BEN WITT KEY, CHAIRMAN.

Dr. Herbert W. Wootton reported a case of **blindness due to optic atrophy,** following removal of the lacrymal gland. The patient, 54 years, developed a circumscribed swelling projecting from under right orbital rim into the tissue of the lid, three months after an attack of grippe. Both breasts had been amputated for carcinoma four years previously. The tumor was removed under local anæsthesia, without accident, and the mass proved to be somewhat hypertrophied, but otherwise normal gland tissue. When the swelling, after operation, had subsided the patient complained that the eye was totally blind. Direct pupillary reaction absent, consensual present. Left eye normal, blood Wassermann negative, and accessory sinuses negative by X-ray. Both fundi normal, with the exception of a guttate choroiditis. Five weeks later, distinct atrophy of the right disk. This case is unfortunately incomplete, inasmuch as the vision of the right eye before operation was not recorded. The patient claims that it was good, and the appearance of the disk, normal at first and subsequently atrophic, would tend to bear out her assertion.

Discussion: Dr. Weeks—I cannot offer any explanation for the cause of optic atrophy, except that in appearance it resembles the atrophy following a sub-periosteal hemorrhage or a fracture through the optic foramen, or post-neuritic atrophy following the attack of grippe, that she had three months previously.

Dr. Walter B. Weidler reported the results of operations

in three cases of **inherited congenital cataracts** extending through four generations. He called attention to the fact that, where a cataract acquired at whatever age, occurs in more than one generation, it descends in the great majority of cases from parent to child, seldom skipping a generation, but it is not uncommon for collateral descendants to be affected also, as far as the fourth generation. In these cases, there were four distinct hereditary defects present, binocular lamellar cataract, lateral nystagmus, microcornea, mental deficiency.

CASE 1.—Catherine Y——, age 19. At age of 16, the right eye was operated upon twice. Cornea measures 9 by 9*mm*, and there is a lateral nystagmus together with slight ptosis of the right upper lid. Vision: O.D. counts fingers eight feet, O.S. fingers eight feet.

CASE 2.—Pearl Y——, age 18. The cornea measures 8 by 10*mm* and nystagmus is more pronounced than in the other two cases. Vision O.D. counts fingers at 1½ feet, O.S. fingers at two feet. A Ziegler discission was performed on the right eye. Seven months after the operation, vision was O.D. $\frac{15}{100}$.

CASE 3.—George Y——, age 7. Cornea measures 7 by 8*mm*, and lateral nystagmus. Vision: O.D. counts fingers ten feet. O.S. counts fingers ten feet. Simple discission was done on each eye twice, resulting vision, O.D. = $\frac{15}{100}$. O.S. = $\frac{15}{100}$.

DISCUSSION: Dr. LAMBERT recalled several cases in one family in which good results were obtained by needling. They possessed one characteristic in that they had some accommodative power which enabled them to read with their distance glasses. In his series of cases transmission occurred through the mother.

Dr. WEEKS called attention to the fact, that in this series, the congenital cataract descended through the males, while retinitis pigmentosa is generally transmitted through the female.

Dr. J. W. SMITH read a paper on **periodic eye examinations** in an industrial plant, in which he showed its great advantage to both employee and employer. The facts presented showed that the employer was able to determine what positions employees could or could not fill, and that subsequent ex-

aminations showed a gradual decrease of abnormalities and eye-strain. Under proper supervision and care, periodic examinations of the eye revealed that no given class of work in a testing laboratory is productive of eye-strain, whether it be clerical or mechanical, or those which involved the testing of very intense light.

Dr. FRANK ALLPORT of Chicago read a paper on **industrial eye accidents.** He divided his subject into two parts; First: Cause and Prevention. Second: Treatment. Under the first caption he called attention to the necessity for examination by an oculist of all applicants for employment, to determine the visual acuity for records in case of accident; to decide what form of work they are best capable of performing, and protect other workers from contagious diseases. Next he called attention to proper illumination, ventilation, and sanitation of shops, the use of protective measures such as goggles and the conditions under which they should be used. The author then took up the management of foreign body invasions of the globe, and stressed the importance of X-ray localization. In extracting he prefers the posterior route, except where the steel is very small or situated anteriorly He then discussed the problem of determining the percentage of loss of vision for compensation; the methods of industrial commissions; and the necessity for a good working scale of visual losses, which the laymen could comprehend. By using a modification of Dr. A. V. Chapman's table, combined with a careful consideration of pathological changes, the author thinks we can arrive at a just conclusion.

DISCUSSION: Dr. SNELL—Snellen notations are not common fractions because they do not partake of the nature of fractions. Each Snellen notation expresses that proportional relationship which is shown by the numerical relation of the apparent numerator and the apparent denominator. The arithmetical series in the Chapman table do not show the exact percentage of loss to physiological visual acuity. Visual efficiency falls by a geometrical ratio. The Chapman series are not mutually proportional whereas the series of test letters in the ratio of progression suggested by Dr. John Green are mutually proportional and measure visual efficiency in a practical way within certain limits. A semi-logarithmic graph

using the 2x ratio will give an approximate percentage of visual acuity efficiency from normal acuity to approximately $\frac{2}{3}\frac{0}{2}\frac{0}{0}$ Sn. but such a graph must assume some point for industrial blindness.

Dr. E. S. SHERMAN: One practical point in making reports on disabilities is the importance of expressing the amount of impairment of function in percentages instead of Snellen notations.

Quite a large proportion of cases of partial loss of vision from industrial injuries is due to infected wounds of the cornea. The worst type of these is the hypopyon or serpiginous ulcer. I have found that the early and thorough application of tincture of iodine to these rapidly spreading ulcers before or just after the formation of the hypopyon, with other suitable local treatment, will almost always check them.

Concerning the route for the extraction of the steel from the vitreous, I believe that small foreign bodies that have entered through the cornea, particularly if the wound of entrance is not closed, should usually be removed by the anterior route. A small magnet such as the Lancaster hand magnet is often very useful, and safer than the giant magnet. Exit of a foreign body through an incision in the sclera is sometimes facilitated by placing a fine silk suture with a very small curved needle in each edge of the wound, and using these as retractors. Another procedure which has been useful when a foreign body refused to come through a scleral incision, is the making of a small cut or nick in one lip of the wound at right angles to the first incision.

Dr. MAY: The subject is very important, especially to those who are called upon for opinions regarding workmen's compensation for ocular disability. I dare say that many ophthalmologists will agree with me that when a man has $\frac{1}{1}\frac{0}{0}$ vision and is awarded 50% of total ocular disability, the award is excessive. An examination of the charts which Dr. Snell has constructed indicates the opposite extreme, that is, an individual with $\frac{1}{1}\frac{0}{0}$ vision is given 10% of total ocular disability; to me this seems too small. I notice that the last proposal of the committee of the Ophthalmological Section of the American Medical Association is to give such a claimant 25% of total visual disability, when vision is reduced to $\frac{1}{1}\frac{0}{0}$; this to my mind seems fair.

Dr. JOSEPH S. SOMBERG: It is true that the Snellen fraction as used is not a true fraction, it is, however, the expression of a distinct proportion which indicates true ratios. These ratios depend upon the size of the letters, the variations in distance, the difference in size of the visual angles, both the complete angle and the minimum separable, and variations in the size of the retinal images.

Dr. BEN WITT KEY: It has been my experience in forming a conclusion as to the loss of visual efficiency of an eye, to look upon the interpretation of the Snellen fractions of visual acuity as one extreme, and the Snell interpretation of these symbols as the other extreme. Everyone knows perfectly well that a patient with $\frac{20}{40}$ vision has not lost, in most cases, $\frac{1}{2}$ visual efficiency of the eye. But has he lost only 10% of vision, according to Dr. Snell's reasoning? My answer to this is, that there are many varieties or grades of $\frac{20}{40}$ vision, each dependent of course upon the character of the opacity or irregularity of the media, error of refraction, or change in the fundus. Since a patient may see $\frac{20}{40}$ very blurred or brilliantly clear in comparison, the correct interpretation of the Snellen fraction in a given case must be expressed in terms of visual efficiency with both extremes as guides to such a conclusion. In other words a man with a large central nebula of the cornea who can with great difficulty make out $\frac{20}{40}$ should receive a greater award than one with a slightly irregular peripheral scar who sees $\frac{20}{40}$ clearly. For the same reason, in an eye with a small irregular dense scar directly over or in the lower quadrant of the pupillary area of the cornea, although $\frac{20}{40}$ plus vision is admitted (but this somewhat blurred), 3 to 5 per cent. loss of visual efficiency may be justly claimed.

Dr. ALLPORT: Dr. Key's remarks are very appropriate. In the making of these tables they all come to about the same when you come to making up the compensation. Whatever table you have, it is only a table, for there are so many things to take into account, besides the acuity. This disagreement between ourselves only makes us the butt of the lawyers in court. The Chapman table is reasonably fair, and when combined with common-sense and judgment, serves the purpose.

MEETING OF DECEMBER 15, 1924. DR. BEN WITT KEY, CHAIRMAN.

Dr. J. S. SOMBERG reported a case of **homonymous quadrant defect** of traumatic origin. A man, aged 45, fell two flights down an elevator shaft striking the back of his head. He was unconscious for four hours. The result of the injury was a right homonymous quadrant defect. The present vision is $\frac{20}{20}$ in each eye. There is a right hemianopsia with preservation of the upper quadrants. As the hemianopic pupillary reflex is absent, Dr. Somberg thinks the lesion is situated in the occipital lobe.

Dr. S. A. AGATSTON presented a case of **retrobulbar gumma** of the orbit. A Chinaman, age 40, six years ago began to have severe headaches in the left parietal region, accompanied by insomnia. Four weeks ago left orbital pain and proptosis with failing vision in the left eye, which showed ophthalmoplegia totalis, exophthalmus, marked increase in intraocular tension, and no perception to light. The fundus showed extensive retinal detachment, and on transillumination was dark. The examination of the abdomen revealed an enlarged liver, and a diagnosis of sarcoma of the liver, with metastatic extension to the orbit, was made. Wassermann was three plus. The X-ray showed diminished illumination in the region of the left maxillary and ethmoidal sinuses. Antiluetic treatment was instituted, followed by rapid improvement. The exophthalmus receded, and the detachment disappeared. There now remains ophthalmoplegia totalis and optic atrophy.

Dr. S. A. AGATSTON also reported a case of **metastatic carcinoma of the orbit.** A woman 40 years of age, who had both breasts removed for carcinoma on August 17th, developed a month later, diplopia, followed by pain and blindness in the right eye. Within a month there was ophthalmoplegia totalis and marked proptosis. The fundus was apparently normal. X-ray of the chest on November 12th, showed that the whole lung was filled with metastatic areas. A re-examination of the fundus at this time showed a thrombosis of the central artery, with a well defined cherry red spot.

Dr. JOHN E. WEEKS read a paper on his travels abroad, in which he visited the principle Ophthalmic clinics in Bordeaux, Paris, Barcelona, Egypt, India, and Japan. He gave a de-

scription of the hospitals, their equipment, and portrayed the operative procedures employed. Having carefully observed the work of many ophthalmic surgeons, and noted that no two men employ exactly the same technique, he concluded that the method of operative procedure employed by men in surgery, should be the one that gives them the best results.

Dr. HARRY S. GRADLE, of Chicago, read a paper on **telescopic spectacles and magnifiers.** In reviewing the literature he showed that this type of aid to vision dates back for more than three centuries, but only of recent years have such become of practical import. The investigations of Hencker and others allied with Zeiss and the urgent need due to the War have resulted in increased usage. Telescopic spectacles consist of a series of lenses, mounted in a case that resembles an inverted jeweler's magnifier and attached to an ordinary spectacle frame, somewhat modified for the weight which is about one ounce. Such a system requires a careful correction of the patient's error of refraction that is incorporated and magnifies the retinal image 1.8. The telescopic magnifier is merely one half of an ordinary theater glass built on the Porros prism principle and modified slightly for the different usage. It magnifies the retinal image from 6 to 30 diameters, according to the system used and is to be employed in cases where satisfactory vision cannot be obtained with telescopic spectacles. There are various devices to enable the user to have free hands for manipulation of the material under consideration. The author then discussed the various cases to which telescopic aids to vision had been successfully applied and pointed out the types to which each form was applicable.

DISCUSSION: Mr. W. B. RAYTON of the Bausch & Lomb Optical Company said Dr. Gradle deserves great credit for his courage and skill in applying these complicated instruments to the improvement of the vision of the unfortunate class of patients he has described. The whole subject is beset with difficulties from end to end, in design and manufacture, in fitting the patient and in the patient's adapting himself to their use. He wished to comment on two or three phases of the subject from the standpoint of an optical engineer rather than from the point of view of an ophthalmologist. It

was surprising to him that Dr. Gradle reports more successful application for near than for distance glasses. The fundamental principle is the enlargement of the retinal image. For near work this can be made as large as we please by moving either head or object so as to sufficiently decrease the distance from object to eye. For distance vision this is impracticable. In the former case, the distance between object and eye may become too short to permit the eye to focus on the object so that some form of aid to accommodation becomes necessary. This can be supplied by a magnifier lens. It seems, therefore, that some of the forms of binocular magnifiers on the market ought to function as well as the telescopic spectacles for reading. For distance nothing but the telescopic system can give assistance. As Dr. Gradle has said, the fundamental optical system consists of an anterior plus element and a negative posterior element, essentially a Galilean telescope. It may be of interest to point out that it is possible to make such a system of a single piece of glass. It becomes too thick and heavy to be practicable. Chromatic aberration is corrected under the assumption that the lenses and the eye are centered on the same axis. Astigmatism at the margin of the field dependent upon the oblique incidence of the pencils of light is corrected for an assumed distance from lens to center of rotation of the eyeball of about 25*mm* allowing 12*mm* from lens to cornea. If the spectacle is not mounted at the correct P.D. or is tilted before the eye both corrections suffer, objects will appear bordered by colored fringes and astigmatism will blur the image. Among other difficulties which the patient is called upon to overcome is that of accustoming himself to the altered space relations. All distances in the line of sight are reduced in proportion to the magnification and those at right angles increased in the same ratio. Aviators refused to attempt a landing with similar spectacles made for them at the request of the government. Accommodation is profoundly affected. For emmetropes, it is reduced by a factor approximately equal to the square of the magnification of the telescope; for hyperopes still more; for myopes less and less. The accommodation with [telescopic [spectacles has been very completely discussed by Dr. Hartinger in *Zeitschrift für Ophthalmologische Optik* for January 25, 1924. The necessity for preserving

the patient's accustomed relation between accommodation and convergence requires a very precise adjustment of the inclination of the axes and a carefully maintained correct working distance if the patient is to get a satisfactory result from a binocular instrument. To secure the harmonious coöperation of two eyes in reading requires much more skill and patience than to successfully equip one eye.

Mr. V. M. E. KOCH, representative of the United States Carl Zeiss agency, said: There is not much more to be said, except for my personal experience in fitting. During four and one half years we have supplied about two hundred telescopic spectacles, one hundred and ten of these since the first of July, 1924. The increased popularity of telescopic spectacles during the last few months is, undoubtedly, due to the work done by Dr. Gradle and Dr. Stein, and to their paper delivered before the American Medical Association at the meeting in Chicago, June, 1924. In stating this number I mean that these spectacles were accepted and retained. There have been failures which I estimate at about 25%, mostly due to the mental attitude of the respective patients. It is a pity that many people requiring such spectacles have not the means to pay for them, and some benevolent persons could do a vast amount of good by taking an interest in this situation. Fitting is difficult and requires the utmost care. After a few weeks the patients should return for adjustment. In many cases telescopic spectacles can be used for distance and for reading. When the visual acuity is too low, telescopic magnifiers may be resorted to. My experience has been that old people do not take very kindly to telescopic magnifiers, probably due to some nervous strain created by their use; but much good can be done with them for younger people, including children. There seems to be an idea that the prescribing and ordering of telescopic spectacles is complicated. This is not so, because all we require is a correct prescription for distance and the visual acuity available. Together with this, correct measurements of spectacle frame should be given, that is to say, interpupillary distance, base and height of bridge and position of crest. If the visual acuity is $\frac{20}{100}$ or better, a pair of telescopic spectacles can be made up from this information. The final pliering should be done by a competent

optician. A great advantage is derived from the possession of a telescopic spectacle trial case, as this enables the prescribing oculist to check up his diagnosis immediately and by trying various lenses arrive at the best possible result. Often one must insist that a patient does not become discouraged in the beginning by the difficulties he experiences, but keep on trying for some weeks. Excellent results are obtained with persons having lack of central vision, but it appears that such cases have sometimes acquired a habit of looking in a slanting way. This habit must be corrected by looking centrally through the telescopic systems. As stated in the beginning, there are about two hundred telescopic spectacles in use in this country. Undoubtedly, a few thousand people could be made more happy and comfortable by the possession of a pair of such sight correcting appliances.

Dr. BERENS: We have seen twelve patients and eight were fitted, six of these comfortably. I have found that a patient must have $\frac{20}{200}$ vision, although in one case the vision was only $\frac{10}{200}$

Dr. DUANE: I had one case which I mention to illustrate some of the points. Woman, seventy years of age, $\frac{20}{200}$ vision with a minus 20. Opacities of the lens. With the telescopic spectacles she could see $\frac{20}{70}$, and with magnifiers read Jaeger No. 1, but on account of the restriction of range and field, she could not carry on her business, and so discarded the glasses. I have no doubt but that in a younger patient under the same condition, the result would have been very satisfactory.

Dr. KEY: Do patients wearing telescopic spectacles complain of very much disturbance of space relations? And does this disturbance disappear after a time? Also has Dr. Gradle observed the effect of the spectacles upon cases of senile degeneration of the macula? The reason I ask this question is because I have seen one case who thought that she had become very much worse soon after trying to wear the spectacles.

Dr. GRADLE in closing said he had not seen any case in which there had been a deterioration of vision following the use of these glasses, nor had he had many complaints about disturbance of space relations. He added that in cases where a patient had equal vision for distance, he fitted a binocular glass for distance, but used one eye only for near.

NOTICE

BRITISH MEDICAL ASSOCIATION

93RD ANNUAL MEETING, BATH, 1925.

President-Elect: F. G. THOMSON, M.A., M.D., M.R.C.P.

SECTION OF OPHTHALMOLOGY.

(*Two Day Section.*)

President: - - WM. MARDON BEAUMONT, M.R.C.S., 4, Gay Street, Bath.

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- ROBERT WALLACE W. HENRY, M.D., 7, De Montfort Street, Leicester.
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- CYRIL HUTCHINSON WALKER, F.R.C.S., 8, Oakfield Road, Clifton, Bristol.

Hon. Secretaries:
- RICHARD COLLEY, M.B., D.O.M.S., 30, The Circus, Bath.
- PHILIP GEOFFREY DOYNE, F.R.C.S., 8, Harley Street, London, W.1.

Feb. 25th, 1925.

DEAR SIR:

The Annual Meeting of the British Medical Association is being held in the historic city of Bath during the week beginning July 20th, and the Section of Ophthalmology meets on Wednesday and Thursday, July 22nd and 23rd.

It is thought, as the Convention of English-speaking Ophthalmologists takes place in London during the preceding week, that there might be some Ophthalmologists from North and South America attending the Convention who would like to visit the Association meeting at Bath.

We are empowered by the Council to say that any such

Ophthalmologists would be welcomed as guests and the Committee would be grateful if you could find space in your Journal to mention this matter. Any further details will be supplied with pleasure by the Secretaries.

We have the honor to be,

Yours very truly,

P. G. DOYNE,
R. COLLEY.